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VOL. 56 MARCH, 1947 No. 1

I

SUBDURAL EMPYEMA SECONDARY TO FRONTAL SINUSITIS

REVIEW OF CLINICAL FEATURES AND REPORT OF THREE CASES

FRANK M. ANDERSON, M.D.

LOS ANGELES, CALIF.

Subdural empyema is not an uncommon intracranial complication of frontal sinusitis, but it is frequently neglected in clinical diagnosis with the result that most cases are discovered at autopsy. The purpose of this communication is to review the clinical picture of subdural empyema in the hope that more of these cases will be recognized early in the course of the disease, when surgery offers a reasonable prospect of cure.

Subdural empyema consists of a collection of purulent material in the subdural space overlying one or both cerebral hemispheres or, rarely, the cerebellum. The subdural space is normally a potential cavity between the dura mater and the arachnoid membrane over the mesial, lateral, and inferior surfaces of the cerebral hemispheres and over the convexities of the cerebellum. Along the line of exit of the

From the Cajal Laboratory of Neuropathology, Los Angeles County Hospital, the University of Southern California School of Medicine, and the Neurosurgical Service, Childrens' Hospital, Los Angeles.

Read before the Western Section Meeting of the American Laryngological, Rhinological and Otological Society, Los Angeles, Calif., January 25, 1947.

cranial nerves at the base, the dura is closely applied to the arachnoid, restricting extension of exudate mesially. At the vertex the pacchionian granulations and the superior cerebral veins entering the superior longitudinal sinus obstruct the space to some extent, but do not prevent mesial and downward extension of pus along and beneath the falx. Over the dorsolateral surfaces of the cerebral hemispheres, the subdural space is not partitioned or interrupted and extends freely from frontal to occipital pole and from vertex to base. Because of these anatomical features, pus in the subdural space is permitted to spread freely and rapidly over extensive areas. When the infection arises in the frontal sinuses, purulent material is most commonly found to extend from the tip of the frontal lobe backward over the frontal, parietal, and upper portions of the temporal lobe. One or both cerebral hemispheres may be covered in their entirety by purulent exudate.

The term "subdural empyema" is preferred to "subdural abscess," at it has been pointed out that a collection of pus in a preformed space should be designated "empyema" rather than "abscess."[1] Other terms which have been used to specify this condition are "pachymeningitis interna," "intradural abscess," "intrameningeal abscess."

This condition may follow otitis media with or without lateral sinus thrombosis[2, 3] and has occasionally been described as complicating ethmoid or sphenoid sinusitis; but the majority of reported cases have followed unilateral acute purulent frontal sinusitis,[4] usually on the same side as the intracranial extension. Subdural infection has been reported following dislocation of a drainage tube in an intracerebral abscess,[5] and metastatic infection of the subdural space from a remote source has been recorded.[1, 6] The exact mechanism of origin undoubtedly varies and has not yet been entirely solved. Direct extension of pus through the posterior wall of the frontal sinus with gross perforation of the dura and subdural invasion is apparently an uncommon sequence. At times osteomyelitis of the frontal bone developing subsequent to frontal sinusitis is followed by extradural abscess, the latter serving as a focus for dural penetration and subdural empyema.

It is probable that infection is in most instances conveyed by way of perforating veins which extend from the frontal sinus mucosa into the dura, since many times at autopsy no gross destruction of the sinus wall or the dura is evident. Courville[7] has pointed out that the process may then proceed with thrombophlebitis of the dural veins, phlebitis with or without thrombosis of the superior longitudinal sinus,

and retrograde thrombosis of the afferent superior cerebral veins. Involvement of the latter vessels would explain development of subdural infections on the opposite side of the brain and at a distance from the infectious source, as occasionally happens. However, it may be that in some cases the subdural exudate is the *cause* of thrombosis of cerebral veins rather than the result.[1] A large percentage of brains with subdural empyema show thrombosis of one or more of these veins; in such instances there are areas of venous infarction in the cerebral substance and, not infrequently, subcortical abscesses. In many cases there is localized meningeal reaction but full-blown septic meningitis is unusual, and the meninges at the base of the brain do not as a rule show evidence of infection. The exudate in the subdural space is fluid in some instances and of a tenacious plastic nature in others. Organization and encapsulation of viscid pus may occur, giving rise to so-called chronic internal pachymeningits, but there is usually little tendency for the more fluid purulent collections to become encapsulated.

Microscopic studies of subdural empyema[8] demonstrate a layer of exudate and organizing granulation tissue adherent to the inner surface of the dura mater and occasionally evidence of neomembrane formation adjacent to the dura. A similar collection of exudate is seen outside the arachnoid, but a neomembrane is not prominent at this surface. Fibrin and leucocytes are observed in the subarachnoid space but organisms are present only in far advanced cases. Softening in the cortex and the white substance of the brain is fairly common and there may be many discrete cortical and subcortical abscesses; thrombosis of cortical veins is a frequent microscopic finding. In very protracted cases, all structures from the dura to the brain substance are united in a dense scar.

For details of pathology and interesting discussions regarding the pathogenesis of subdural infections, reference should be made to the excellent articles of Courville,[9, 10] and of Kubik and Adams.[1]

Subdural empyema has been most frequently reported as complicating acute rather than chronic frontal sinusitis, though 8 of 12 cases reported by Kubik and Adams[1] developed after an exacerbation of chronic sinus disease. They believe that chronic frontal sinusitis with frontal osteomyelitis and epidural infection is particularly favorable to the development of subdural empyema. The course of the disease is frequently short, with a survival period at times of only a few days. Isolated and acute unilateral frontal sinusitis associated with local tenderness, swelling, and redness is followed almost imperceptibly by signs of intracranial extension. Advancing infection is

indicated by irritability, frontal or generalized headache, malaise, fever with occasional chills, vomiting, leukocytosis, and signs of meningeal irritation. Lethargy and progressively deepening stupor are invariably observed within a few hours or days. As the subdural exudate accumulates and venous thromboses occur, the intracranial pressure rises; by this time, lateralizing or focal signs are usually evident. These include: hemiparesis, flaccid or spastic, most frequently contralateral to the brain lesion; Jacksonian or generalized convulsions; motor aphasia; and inability to deviate the eyes conjugately to the opposite side. Sensory impairment and visual field defects are rarely demonstrable, perhaps because of stupor and limited cooperation, but if carefully looked for, minor differences in the deep and superficial reflexes or the presence of Babinski signs can frequently be shown, and these reflex findings may constitute the only positive neurologic signs. Papilledema is often detectable and dilatation of one pupil is occasionally seen; the larger pupil may, however, be on the side opposite the empyema.

Spinal puncture reveals increased pressure and elevated protein with mild to moderate pleocytosis. High cell counts have been reported[3, 11] but are unusual. In most cases no organisms have been cultured from the spinal fluid but growth may be obtained, and such a finding must not make one conclude that the case is one of meningitis without the possibility of subdural empyema; the two states may co-exist or the organism may merely be a contaminant.

In almost all cases the clinical condition becomes increasingly more critical without remission and septic meningitis frequently supervenes in the terminal stages. Unless drainage is provided, death usually occurs within a week after the onset of lateralizing signs. In rare instances the subdural pus becomes organized with subsidence of acute symptoms and the establishment of "chronic pachymeningitis interna," a state which may eventually resolve with clinical cure.[12]

Diagnosis of subdural empyema should not be unduly difficult if one is cognizant of the usual course followed by this condition and realizes the possibility of its existence in the case at hand. Frequently this complication is not even considered when diagnostic possibilities are enumerated. In acute cases of frontal sinusitis with or without antecedent sinus disease, followed by orbital swelling, persistent headache, malaise, fever at times with chills, meningeal signs, and lethargy, the observer should be very suspicious of subdural empyema. The appearance of focal or generalized convulsions, hemiparesis, aphasia, and deviation of the eyes almost assure the diagnosis and indicate that surgical drainage is imperative.

Differential diagnosis of the well-advanced case rests between intracerebral abscess and longitudinal sinus thrombosis. Ordinarily, the early stage of intracerebral abscess formation is by comparison milder and more protracted. The headache tends to be less severe and to vary considerably in intensity, the temperature is not remarkably elevated, and meningeal signs are absent. There are times, however, when intracerebral abscess forms rapidly and creates a clinical picture much like subdural empyema. In such a case, a diagnostic burr-hole in the frontal region will settle the issue. Thrombosis of the longitudinal sinus produces a clinical syndrome similar to that of subdural empyema and, as above mentioned, these states may coexist.[13] Differentiation may be very difficult, but in thrombosis one is more likely to find alternating convulsions right and left, often beginning in the foot; bilateral signs, such as weakness of both legs; a septic type of temperature curve; and not infrequently, a variable course with remissions.[14] Meningeal signs ordinarily are not prominent in this syndrome and the spinal fluid is free of cells.

Purulent meningitis, particularly when accompanied by thrombosis of cortical blood vessels, may offer confusion in diagnosis, although this condition is rarely an uncomplicated sequel of frontal sinusitis. The course of meningitis is as a rule much more gradual and the picture less dramatic than in subdural empyema; careful clinical observation and spinal fluid study should afford differentiation. Extradural abscess complicating frontal sinusitis gives rise to a much milder illness than does subdural empyema. With extradural abscess there are no signs of meningeal irritation, no focal signs of cerebral damage, and the patient does not appear critically ill. In a patient with increased intracranial pressure, stupor, meningeal signs, and focal neurologic findings, the discovery of extradural pus should not delude one into believing this to be the only complication; subdural empyema very likely is present in addition to the extradural abscess.

Subdural empyema is prevented from developing in most cases of frontal sinusitis by active treatment of the sinus, including trephination when indicated and administration of the sulfonamides and penicillin. When the subdural exudate is established, however, the only effective treatment is surgical drainage.[1, 3, 11] This is best accomplished through strategically placed burr-holes arranged to allow thorough irrigation of the subdural space. Because the exudate is usually most copious in the frontal area, the first burr-hole should be placed in the midlateral frontal region near the coronal suture on the side opposite the Jacksonian convulsions or the hemiparesis. The opening is enlarged moderately with a rongeur and the pale tense dura

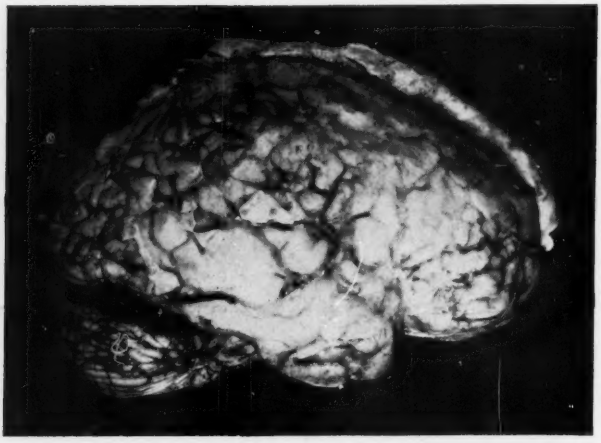

Fig. 1. (Case 1.)—Photograph showing extensive subdural empyema overlying most of the dorsolateral surface of the right cerebral hemisphere. Much of this thick, tenacious exudate remained adherent to the dura as can be observed in Fig. 2.

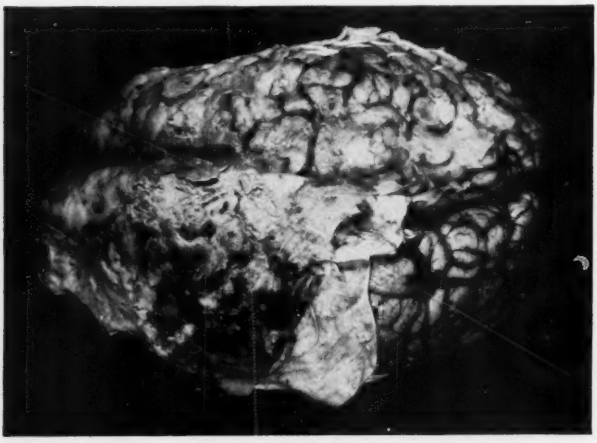

Fig. 2 (Case 1).—Dorsal view with reflected dura mater exposed to show massive accumulation of viscous pus on the under surface of this membrane. There is indentation of the right frontal lobe, caused by pressure of the overlying exudate.

is opened with a cruciate incision, care being taken not to injure the arachnoid. By carefully retracting the brain and irrigating the subdural space with saline, the purulent material in this area is evacuated as thoroughly as possible. An opening is then made in the parietal region and perhaps also in the temporal region, and through-and-through irrigation is carried out. If it is evident that pus extends beyond the accessible limits of these openings, additional burr-holes are inserted and the irrigation continued. Often it will be found advisable to make an opening in the opposite side as well, to exclude bilateral empyema.

Penrose drains are placed in several directions in the subdural space, penicillin solution instilled, and the scalp wounds loosely closed; the dura is not sutured. The drains are shortened in two or three days and removed in about six days. Sulfonamides and penicillin are continued until the patient is afebrile and the scalp wounds have healed. Careful attention to the general care is highly essential, since these patients are critically ill and in need of competent nursing, nutritious diet, blood transfusions, and similar supportive measures.

REPORT OF CASES

CASE 1.—(L.A.C.H. autopsy No. 32331.) G. N., a 16-year-old girl, was admitted to the Los Angeles County Hospital on April 15, 1944. Three weeks before she had developed a respiratory infection after swimming and had gone to bed with fever and malaise. There is no record of treatment for sinusitis but an adequate history was never obtained. On admission the child was lethargic and her temperature was 101.4° F., but she had no symptoms referable to the sinuses and there were no direct evidences of intracranial disease. The fever continued and a week later the patient rather abruptly complained of headache and stiff neck, at which time spinal puncture disclosed pressure of 400 mm., 3 plus Pandy test, and 46 lymphocytes. Spinal fluid culture yielded hemolytic streptococcus, and sulfadiazine administration, 1 gm. every four hours, was begun immediately. The following day a flaccid left hemiparesis appeared with increased deep reflexes and a left Babinski sign; a left peripheral facial palsy, engorgement of the retinal veins, and pronounced meningeal signs were also described. Mastoid x-ray films were interpreted as showing chronic mastoiditis on the left with acute infection on the right. The next day (April 26) there were several generalized convulsions, most prominent in the left extremities, suggesting a diagnosis of septic meningitis with cortical thromboses, possibly secondary to mastoiditis. Her condition became progres-

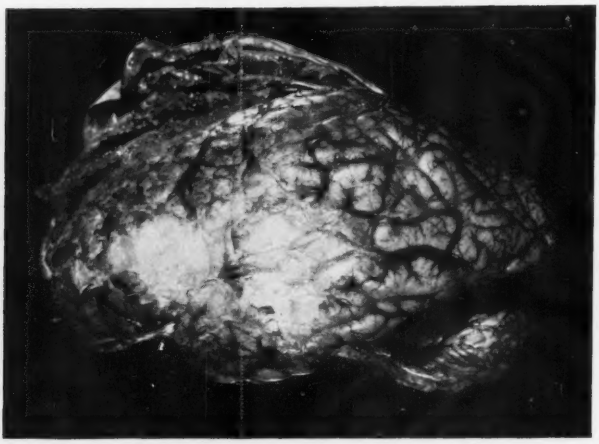

Fig. 3 (Case 2).—The subdural empyema in this case extended posteriorly from the left frontal pole and covered portions of the parietal and temporal lobes, but a considerable part of the rather fluid pus was lost in fixation. A collection of more viscid exudate is visible in and adjacent to the Sylvian region. A smaller subdural empyema was present over the opposite frontal lobe.

sively worse and the patient died April 28, about five weeks after onset of the illness.

Autopsy disclosed a bilateral frontal extradural abscess, 8 x 10 cm., more on the right side; marked thickening of the dura in the right frontal region (0.5 cm.); and an extensive collection of subdural pus over the entire dorsolateral surface of the right cerebral hemisphere with marked depression of the brain and small scattered collections of purulent exudate in the subarachnoid space (Figs. 1 and 2). No venous thromboses were noted, and there were no intracerebral abscesses. The right frontal sinus was filled with thick, yellow-green, purulent material similar to that in the subdural space, but there was no perforation of the posterior sinus wall or the dura, and all other sinuses and both mastoid bones were normal. A hemolytic streptococcus was cultured from the subdural pus.

Histologically the subdural and extradural exudates were found to be partially organized and intimately adherent to the thickened dura. The dura was freely infiltrated with polymorphonuclear leucocytes and lymphocytes. Diagnoses: bilateral frontal extradural ab-

scess; right subdural empyema; minor localized meningitis; all secondary to purulent right frontal sinusitis.

CASE 2.—(L. A. C. H. autopsy No. 36261.) The patient (S. W.) was a 28-year-old negro male who had sustained a minor head injury one year before and had complained of headache and "sinus trouble" since that time. He had been treated for lues some years before and his blood Wassermann had become negative. On October 4, 1946, he developed severe headache (location not stated), fever, and had two chills. The headache and fever continued and on the third day the patient became stuporous. He was admitted on October 7 with right hemiparesis, motor aphasia, meningeal signs, stupor, and fever of 101.8° F. The spinal fluid pressure was 250 mm. and the fluid showed 360 cells per cubic mm., 85% of which were lymphocytes; staphylococcus aureus and albus were cultured from the fluid. The spinal fluid Wassermann reaction was negative, the blood Wassermann negative and the Kahn test doubtful. Tentative diagnoses included poliomyelitis and syphilitic meningitis. Fifty thousand units of penicillin were administered every three hours and 1 gm. of sulfadiazine every four hours.

The following day there were several Jacksonian convulsions involving the right extremities, the left pupil became dilated, the right superficial abdominal reflexes could not be obtained, and Babinski signs were positive bilaterally. No papilledema was observed. On this date the spinal fluid cell count was 518 with 78 per cent polymorphonuclear cells, and although no organisms could be cultured, meningitis of undetermined type, possibly tuberculous, was thought to be the most likely diagnosis. Three days before the patient's death, a definite history of chronic frontal sinusitis was obtained from relatives and the diagnosis of subdural empyema was considered. The patient's condition was very critical, however, and an operation was not performed. Death occurred October 14, 1946.

Autopsy disclosed thick yellow pus in both frontal sinuses and a small (4 x 1.7 cm.) midline extradural abscess at the frontal poles but no gross perforation of the sinus walls. There was a 1 cm. necrotic spot in the dura at the right frontal pole and thick yellow purulent material was found in the subdural space overlying the anterior portion of the right frontal lobe. An extensive collection of subdural pus was found over the dorsolateral aspect of the left frontal lobe, a portion of the parietal lobe, and the upper half of the temporal lobe (Fig. 3), the empyema on this side being much more abundant than on the right. The sagittal sinus showed no throm-

boses, though scattered thrombi were observed in the superior cerebral veins.

Microscopic sections closely resembled those in Case 1, except that meningitis was more pronounced subjacent to the empyema and inflammatory reaction could be noted in the cortex at scattered points. Venous thromboses were distinct. Anatomic diagnoses: suppurative frontal sinusitis, bilateral; epidural abscess, adjacent; subdural empyema, bilateral; secondary meningitis. Staphylococcus aureus and a gamma streptococcus were cultured from the subdural pus.

CASE 3.—(Childrens' Hospital No. 45-4250.) The patient, a 13-year-old girl giving no antecedent history of sinusitis, developed fever, pain and tenderness in the region of the left frontal sinus and purulent drainage from the left nostril a few hours after swimming in a public pool, October 1, 1945. Her temperature rose to 102° F. and continued near this level for a week, when it abruptly rose to 105,° at which time the patient vomited several times and complained of severe malaise. She was hospitalized elsewhere; nasal shrinkage was instituted, and sulfadiazine, penicillin, and blood transfusions were administered. X-ray studies demonstrated clouding of the left antrum but the other sinuses appeared clear. The general condition did not improve and on October 21, three weeks after onset of the illness, the patient had a generalized convulsion, became stuporous, and developed difficulty in speech and weakness of the right arm. On October 23, 1945, she was admitted to Childrens' Hospital.

Examination on admission showed the child to be stuporous but able to cooperate. Tenderness without swelling was present over the left frontal sinus and questionable papilledema, marked motor aphasia, and impairment in recognition of objects were observed. No visual field defect was made out and examination of the ears was negative. Mild paresis was noted in the right arm, but movements of the face and legs were normal and sensation was considered to be intact. Minimal meningeal signs were present, the deep reflexes in the right extremities were slightly increased with a right Babinski sign, and the superficial abdominal reflexes were diminished on this side. The spinal fluid contained no cells, 30 mg. of protein, and 60 mg. of sugar; the presure was not recorded but puncture the following day disclosed a pressure of 325 mm., and 33 cells, including many red blood cells.

Operation was performed on the day of admission. A burr-hole was placed in the left frontal bone at the hairline and enlarged

slightly with rongeurs. When the dura was cautiously incised, a gush of thin yellow pus issued from the subdural space. Burr-holes were inserted in the left parietal and temporal areas, in both of which subdural pus was encountered. By carefully retracting the brain, a considerable amount of thin, unorganized pus was evacuated. Through-and-through saline irrigation of the subdural space was then carried out. Penrose drains were placed through each of the burr-holes into the subdural space and the scalp wounds were loosely closed.

Fifty thousand units of penicillin were given intramuscularly every three hours and 15,000 units were instilled intrathecally twice a day for three days, then daily for four days; 1 gm. of sulfadiazine was given every six hours. Staphylococcus albus was cultured from the subdural pus and from the nasal discharge, and on one occasion this organism was cultured from the spinal fluid, though two other spinal fluid cultures were negative. For 12 hours following operation there was little change in the patient's condition, but at that time steady improvement began and by the fifth postoperative day speech was almost normal and no weakness of the arm was discernable. The drains were removed on the sixth postoperative day and the patient was allowed to be out of bed 10 days later. She was discharged from the hospital on the 24th postoperative day and has remained entirely well and free of symptoms (16 months).

Comment. In the first case a complete history was not recorded and it has not been determined whether the patient suffered from frontal sinus infection prior to her last illness. It is clear, however, that she presented no symptoms or signs indicating acute sinusitis and the clinical findings were not ascribed to complications from this source. Apparently the purulent right frontal sinusitis gave rise to no local symptoms yet was the source of an extensive subdural empyema on the same side, and since there was no perforation of the sinus wall or the dura, it is presumed that the infection was carried by venous channels. Although the mastoid x-rays were thought to show evidence of infection, autopsy disclosed no trace of acute or chronic disease in the temporal bones.

The second patient might have been saved had the history of frontal sinusitis been clearly defined and fully appreciated before his condition became hopeless, because the clinical picture was classical of subdural empyema. The issue was confused by the history of syphilis and by the account of chronic headache following a minor head injury. Also, the patient was admitted to the hospital at the

time of a poliomyelitis epidemic. Because at autopsy both frontal sinuses were found to be infected, it is not possible to say which of these constituted the source of the bilateral subdural empyema. Although there was a necrotic spot in the dura at the tip of the right frontal lobe, the subdural pus was much more abundant on the left, suggesting that infection may have travelled through the dural veins on the right into the longitudinal sinus (but without thrombosis of the sinus), then into the superior cerebral veins on the left.

The result in the third case can be credited to the fact that operation was performed relatively early, before irreparable brain damage occurred. It is easily possible that delay of another 24 hours would have resulted in serious cerebral damage due to venous thrombosis and direct pressure from the accumulated pus. In such an event, one might expect greatly protracted convalescence with persisting convulsive seizures, hemiparesis, or other disturbing sequelae.

CONCLUSIONS

1. The subject of intracranial subdural empyema (subdural abscess) complicating purulent frontal sinusitis is reviewed and three cases of this syndrome are presented.

2. Subdural empyema may follow the initial attack of frontal sinusitis or it may appear during an acute recrudescence of chronic frontal sinus disease. Otitis and sinus infections other than frontal are less common sources of infection in the subdural space, and origin by metastasis from a remote source is rare.

3. The mortality from subdural empyema has been high, primarily because this lesion is not considered in diagnosis. The appearance of headache, stiff neck, somnolence, Jacksonian convulsions, and hemiparesis in a patient with frontal sinusitis renders this diagnosis very probable.

4. It is imperative that drainage be provided as soon as the diagnosis is reasonably certain. This is best accomplished through enlarged burr-holes in the frontal, parietal, and temporal regions, so arranged that thorough irrigation of the subdural cavity can be carried out. Drains are maintained in the subdural space for four to six days following operation.

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II

MÉNIÈRE'S DISEASE

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Ever since Ménière[27] drew attention to the labyrinth as the seat of the disorder responsible for severe vertigo accompanied by unilateral deafness, his name has been attached to this syndrome and under this umbrella term a variety of otological conditions, irrespective of their pathology, has been allowed to shelter.

It has long been appreciated that, after excluding inflammatory, neoplastic and posttraumatic conditions of the labyrinth and the eighth nerve, there still remains a large group of cases characterised by paroxysmal vertigo with nausea and vomiting, deafness and often tinnitus, in which no other neurological changes can be demonstrated.

The nonfatal course of this condition has prevented the accumulation of pathological evidence as to the true nature of the disorder and it was not until as recently as eight years ago that a specific disease process within the labyrinth was identified.

This condition, named by general consent Ménière's disease, is the subject of this communication, the clinical details of which are based upon personal experiences gained during the investigation and treatment of over 400 cases.

Paroxysmal vertigo is no new condition for in the early days of the Christian era in Rome when it was the custom to assign diseases of certain systems or groups of symptoms to a patron saint, vertigo was placed under the exclusive patronage of a certain Saint Avertin.[17] Upon further investigation into this, Guthrie,[18] suggests that no such saint really existed, but there can be no doubt that vertigo was a common disturbance even then.

Among the historical figures who are believed to have suffered from Ménière's disease was Martin Luther. To a man who complained to him of the itch, Luther said: "I would give ten florins to change with you; you know not how distressing vertigo is. At

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this very moment I am unable to read a letter through at once, and indeed I cannot read more than two or three lines of my psalter; for when I make the attempt such a buzzing comes on in my ears that I am often on the point of falling off my seat. On the other hand the itch is a useful thing"[28]

Dean Swift was also tormented by giddiness and was finally committed to a madhouse. In 1725 he ended a letter to Pope with, "This wildness you must allow because I am so giddy and deaf." Despite the attention lavished by the doctors upon his digestive tract and the frequent blood lettings, Swift stubbornly and, as it has turned out, shrewdly maintained that the cause of his illness lay within his skull and not in his blood nor yet in his belly.[24]

Ménière's original paper, in which he described the postmortem findings in the case of a young woman who died after several days of continuous vertigo and unilateral deafness, has been criticised, particularly by McKenzie,[26] Dederding[11] and more recently by Atkinson.[4] Quite clearly Ménière was so determined to confirm his theory that the ear was responsible for the bouts of vertigo that he grasped eagerly, it might almost be said a shade too eagerly, at the opportunity presented by this unfortunate girl's death. From the course of the illness it would seem likely that the condition was due to infection or hemorrhage of leukemic origin, neither of which we now consider play any part in the disease which bears his name. Nevertheless it was Ménière, through this paper, who was responsible for directing the attention of clinicians to the labyrinth as the seat of the disorder.

Over 50 years ago Knapp and Cheatle[22] put forward the suggestion that the cause of the disease was an increase of pressure within the labyrinth, and compared the condition with glaucoma. Many other observers thought along similar lines and Dederding[11] gave an excellent picture of the condition and drew attention to the part played by fluid metabolism in the production of the symptoms. Furstenberg, Lashmet and Lathrop[15] suggested that retention of sodium within the body was the responsible factor, and considered fluid metabolism to be of secondary importance. Wright[33] and Crowe[9] each contributed valuable clinical descriptions of the disease and each emphasized the possibility of some disturbance of endolymphatic function, either physico-chemical (Crowe) or inflammatory (Wright) as the cause. Shortly after this, Hallpike and Cairns[20] published the result of a histological examination of the temporal bones in two cases of Ménière's disease. In each case, on

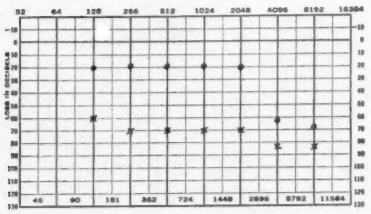

Fig. 1.—The above audiogram was made in a quiet room with a Western Electric 2 B. audiometer. Hearing loss for air conducted pure tones is denoted by "o" for the left ear and "x" for the right ear.

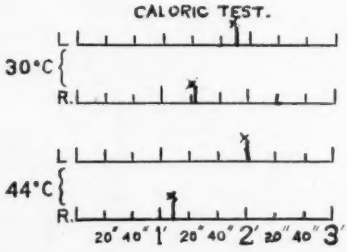

Fig. 2.—The above chart is a convenient and pictorial way of setting down the result of the caloric test that has already been described,[14-19] and will later be referred to in the text. The method has been used in all the cases in the present series and is carried out as follows:

With the patient lying supine, the head is raised forward by 30° to bring the lateral semicircular canal into the vertical plane.

Water from a two-pint douche-can fixed about two feet above the level of the patient's head is led to the ear through about six feet of rubber tubing, and allowed to flow for exactly 40 seconds, the diameter of the outflow nozzle being such that not less than eight ounces flow through during the 40 seconds, the average amount being from 12 to 14 fluid ounces.

Each ear is thus douched in turn with water at exactly 30° C. and then with water at exactly 44° C., the temperature being measured in the douche-can immediately before the flow is started. At least five minutes' pause is allowed between each syringing.

During and after each test the patient is instructed to fix his gaze upon some relatively distant object straight ahead of and slightly above him, so that the eyes are voluntarily held in the midway position.

With good illumination any involuntary eye movements that result from the syringing are observed and the duration of any after-nystagmus noted in minutes and seconds from the onset of the stimulus to the end-point of the nystagmus.

the affected side only, there was found to be an extreme dilatation of the endolymphatic system and changes in the absorption area of the endolymph around the saccus endolymphaticus. Nothing to suggest an inflammatory cause for these changes was noted. It is interesting to note that these three important papers, the first two largely clinical and the third pathological, were all published within the space of a few months. Similar findings were recorded in another case by Hallpike and Wright,[21] in six cases by Rollin,[30] in a case by Lindsay[25] and in three cases by Altmann and Fowler.[2]

Pathology. Figures 3-8 were prepared by Hallpike from the temporal bones of a case of Ménière's disease. The subject was a man, aged 46, who for four years had suffered from sudden bouts of giddiness. Sometime after the onset of these bouts he noticed deafness of the right ear.

On examination in March 1942 there was not any evidence of oronasal infection and the tympanic membranes appeared normal. Tests of cochlear and vestibular function revealed a severe perceptive type deafness on the right side and on caloric stimulation there was a deficiency of the right responses as compared with the left.

As the patient did not respond to conservative treatment he was recommended for labyrinthectomy. Whilst awaiting admission to the hospital he developed pallor and became very weak and when admitted in December 1942, the blood examination revealed an acute lymphatic leukemia and death ensued eleven days later. There is no evidence of intralabyrinthine hemorrhage in this case, and there is no reason to suppose that there was any connection between the two diseases. It will be noted that the essential features seen in previous cases, namely distension of the cochlear canal and changes in the perisaccal connective tissue, can be seen on the affected side in these sections.

It may therefore be said that a firm pathological basis for the disease has been established and that the essential mechanism of the disorder is an obstructive distention of the endolymphatic system. According to Guild[16] the fluid filling the endolymphatic system is derived from the stria vascularis and passes through the wall of the pars intermedia of the saccus endolymphaticus into the loose perisaccal connective tissue. Aldred, Hallpike and Ledoux[1] made measurements of the osmotic pressure of the labyrinthine fluids and their findings suggest that the endolymph must be pumped out of the saccus through the vital activity of the cells lining its wall and once

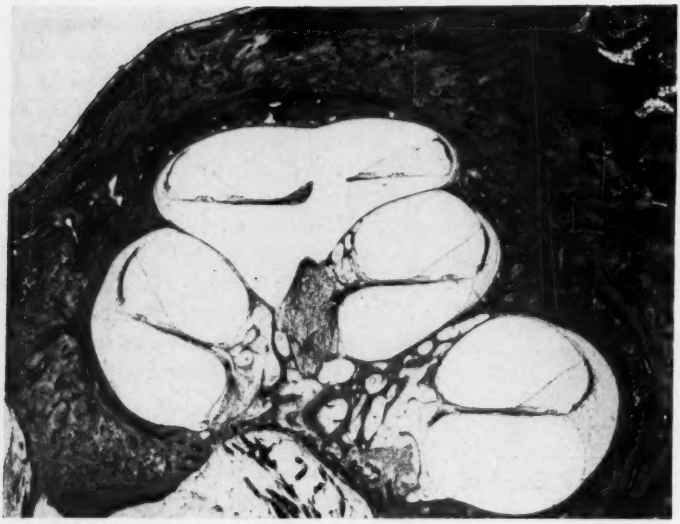

Fig. 3.—Mid-modiolar section of the cochlea on the unaffected side. (x13)

in the meshes of the perisaccal connective tissue be diluted by water attracted to the osmotically stronger endolymph; it is then absorbed into the blood stream. The changes in the perisaccal connective tissue that have been noted in cases of Ménière's disease would be sufficient to interfere with this delicate process, with the result that the endolymphatic system could not adjust itself adequately to any sudden variations in tension to which it might be subjected. Given, therefore, some delay in the disposal of surplus endolymph, it is possible that a variety of factors may be responsible for a sudden increase in endolymphatic pressure, sufficient temporarily to interfere with normal vestibular activity, thus precipitating an attack.

Mygind and Dederding[29] postulate a general disorder of water metabolism, whilst Dohlman,[12] Atkinson[3] and Williams[32] favour an allergic reaction. Wright[33] considers that a focus of sepsis may be responsible, drawing the analogy between this condition and the increased intraocular tension in iritis. In the series of cases under review, however, very few instances of probable foci of sepsis were encountered.

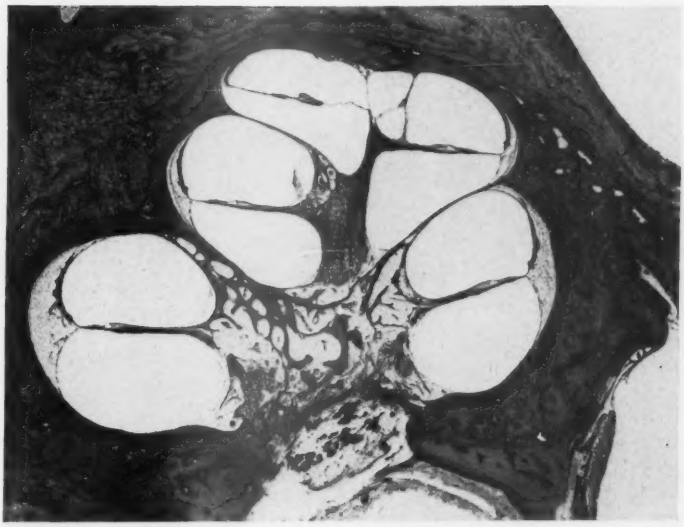

Fig. 4.—Mid-modiolar section of the cochlea on the affected side. Reissner's membrane has been pushed back against the bony wall of the scala vestibuli so that it cannot easily be seen except where it is blocking and bulging through the helicotrema. (x13)

No matter what the exciting cause may be, some holdup in the emptying of the saccus must be present before the factors mentioned can operate.

It is felt that undue attention has in the past been paid to the influence of eustachian insufficiency and other conditions of the middle and even external ear upon Ménière's disease. Given a relatively closed endolymphatic system, it is conceivable that sudden and comparatively gross movements of the ossicular chain might precipitate an attack, but the very small number of cases in which any abnormality of the external ear or the middle ear cleft has been discovered makes it difficult to believe that the conducting part of the auditory apparatus can often be held responsible for the attacks. The pathological picture, taken in conjunction with the absence of any signs of disease within the central nervous system and the relief from attacks following destruction of the end organ, is not in accord

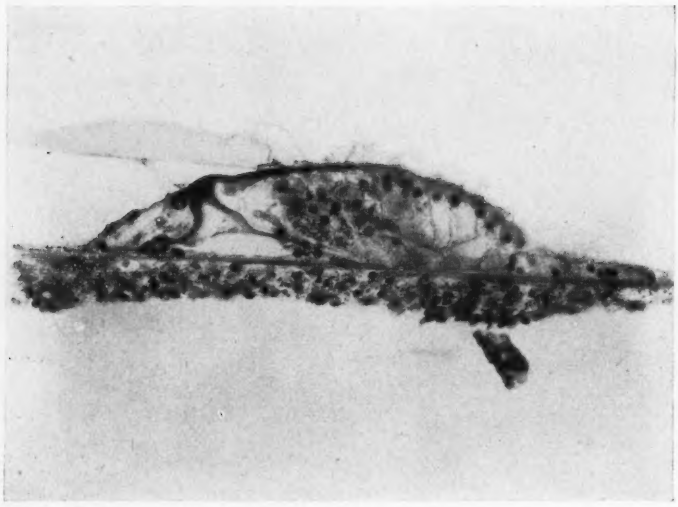

Fig. 5.—The organ of Corti on the unaffected side. (x320)

with Dandy's[10] view that these cases are often due to pressure upon or changes within the trunk of the eighth nerve.

In none of the present series of cases has there been a history of former head injury, and although such injuries are frequently followed by symptoms and signs of labyrinthine disturbance,[6] the clinical features of the two conditions are usually quite distinct and separate.

In the light of present knowledge, Dederding's[11] work on fluid retention, and Furstenberg, Lashmet and Lathrop's[15] modification of this deserve careful attention, because, in the presence of a relatively closed endolymphatic system, variations in fluid metabolism may easily affect the endolymph pressure. Cawthorne and Fawcett[7] found in a small series of cases that striking variations in the symptoms could be caused by preventing or encouraging fluid retention.

Clinical Features. Of the 424 cases under review it will be noted in the following table that slightly more than half were in males; whilst three-quarters occurred between the ages of 30 and 60.

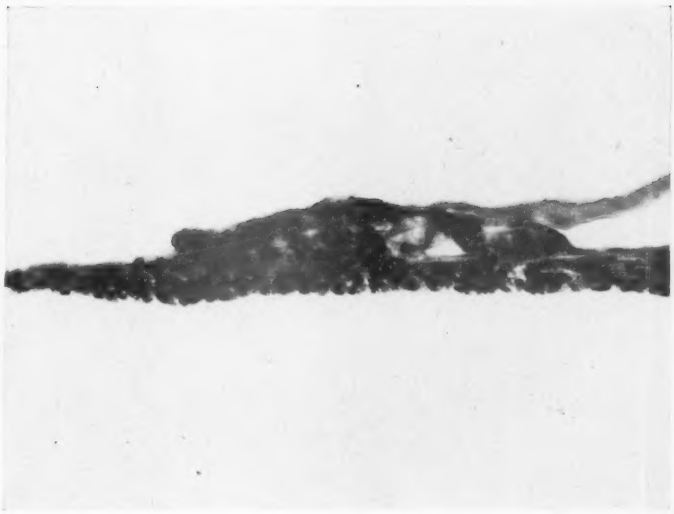

Fig. 6.—The organ of Corti on the affected side. It is obviously compressed and the dark staining has been attributed by Hallpike & Cairns[20] to an anoxic condition of the cells brought about by pressure. (x320)

MÉNIÈRE'S DISEASE: SEX AND AGE INCIDENCE

TOTAL	424
MALE	234
FEMALE	190

AGE-GROUP	NUMBERS
10 - 20	6
20 - 30	27
30 - 40	99
40 - 50	135
50 - 60	92
60 - 70	52
70 - 80	12
80 and over	1

The general picture of the disease is highly characteristic and was exhibited by a large proportion of the cases under review, though the signs and symptoms were found to vary in their relative intensity and in the order of their first appearance.

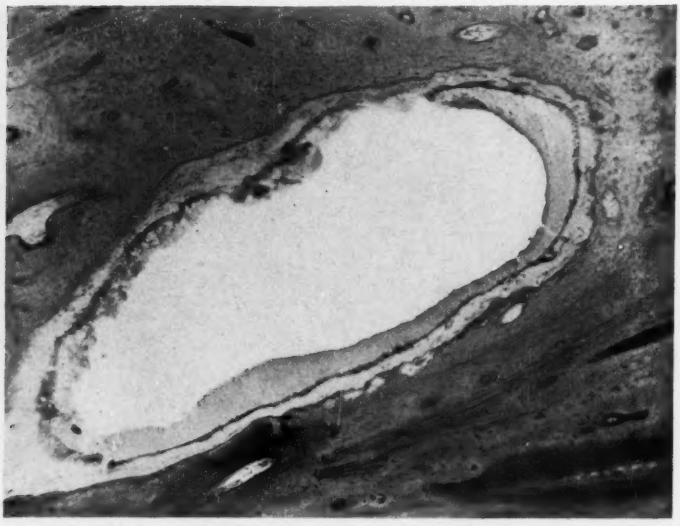

Fig. 7.—The saccus on the unaffected side. (x88)

In all cases there were, for no apparent reason, periodic bouts of paroxysmal vertigo accompanied as a rule by nausea and vomiting, deafness of the perceptive type, always more noticeable on one side, and tinnitus, but no other signs of disease within the central nervous system. All these accompaniments of the vertigo together with other less constant features will be commented on seriatim. A variable and sometimes distressing feature of the complaint was the depressing influence that these sudden attacks exerted upon the general well-being and everyday life of the patient. This can well be illustrated by the retort of the patient who when chided for having given way too much replied, "Well doctor, if you found yourself suddenly and without warning in the middle of an earthquake being horribly seasick, wouldn't you feel sorry for yourself, and wouldn't you dread the thought of further attacks?" Obviously to withstand these attacks with equanimity calls for a stout heart, and it is not surprising that one encounters a variety of psychological disturbances as the result of repeated attacks.

The nature, intensity, direction and duration of the attacks varied considerably, not only from patient to patient but also at

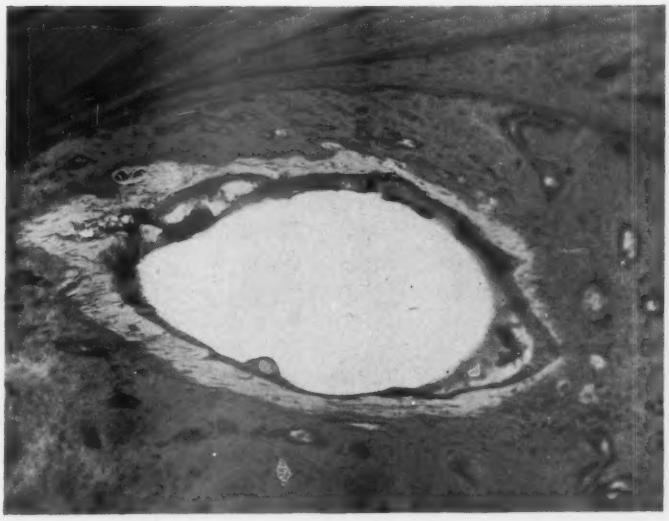

Fig. 8.—The saccus on the affected side. On both sides there is no loose perisaccal connective tissue, and the epithelium is backed by dense fibrous tissue lining the bony wall of the cleft. This structural peculiarity has been noted in a proportion of normal individuals as well as those suffering from Ménière's disease,[20] and this may account for the abnormality being present on the unaffected side. (x88)

times from attack to attack. The bouts tended to occur in groups, there being several within a relatively short period, followed by a long interval of freedom sometimes extending over years.

Much of what follows will refer to severe attacks, but it has been found that in some patients the attacks were always slight, in others always severe, whilst in others there was a haphazard mixture of slight and severe attacks.

Attacks did not appear to have been precipitated by any particular activity with the possible exception of early morning attacks shortly after waking or rising. They were not, however, respecters of time or of activity and even happened at night. In fact most patients stated that occasionally they were woken up by an attack during the night.

In somewhat less than half of the cases there was warning of an impending attack. An increase in the intensity, or an alteration

in the character of the tinnitus was often noted before an attack, whilst sometimes there was in addition an accentuation of the deafness or an increased dislike of loud sounds. A feeling of fullness, distention, or numbness in or behind the affected ear was also noted, whilst as a variant, a headache, usually described in terms of pressure or bursting rather than pain, was sometimes experienced. Most of the warning symptoms continued throughout the attack and often lasted for a short while afterwards. In some instances there was a feeling of lightheadedness and slight disturbance of balance. These premonitions were usually experienced only a short time before the attacks, but in a few instances the aura preceded the attack by a day or even longer. It was found that when the attacks occurred in well-defined groups with long intervals of freedom, the aura preceding a group of attacks might persist until the attacks had worked themselves out.

The attack usually took the form of a sensation of movement of surrounding objects, less commonly of the subject himself and but rarely a combination of both. The direction of the apparent movement could sometimes be described but frequently the turbulence of the sensation so affected the sufferer that an accurate description was impossible. Attacks might last a matter of minutes or a few hours and, if severe, might render the patient helpless. Certain determined patients could, however, if an attack of not more than moderate severity overtook them in the street, sometimes find their way home before collapsing. One patient had an attack whilst driving his car alone; he was able to drive home and into his garage but had to be lifted out of the car as he was quite unable to walk.

In many instances, following a sound sleep of several hours, the patient woke little the worse for his attack. In other cases the effects wore off more slowly but they rarely lingered more than a day or two.

Nausea and vomiting usually accompanied or followed severe attacks. The tendency for vomiting to appear towards the end of an attack is no doubt responsible for the widely held belief, sometimes amounting to an unshakable conviction, that a digestive disturbance is the cause of the trouble. The accompanying pallor, cold sweat, slow thready pulse and increased activity of the stomach and biliary passages and sometimes even of the intestinal tract all point to a strong vagal storm provoked by impulses spreading to the vagal centres from nearby vestibular nuclei.

These two symptoms of a cochlear disturbance, deafness and tinnitus, usually went together and they were rarely absent in the established disease. The deafness in Ménière's disease was of the perceptive type and it presented two distinguishing features to which particular attention is drawn. In the first place the deafness was subject to sudden variations, possibly as the result of changes in endolymphatic pressure and this was particularly noticeable before the cochlea, as the result of severe or repeated attacks, had lost its resilience. This variability of the hearing has been adduced in favour of a conductive deafness, and it is not always appreciated that quite wide fluctuations in hearing can occur in perceptive deafness: it seems reasonable to consider that they are due to changes in endolymphatic pressure. The second, and as will be shown later, an important feature of this type of deafness was the discomfort due to distortion of loud sounds, particularly when they were high-pitched. Music heard over the radio, children's voices and the rattle of crockery often jarred horribly, especially during the cycle of an attack. Other loud sounds, not necessarily high-pitched, were apt to cause distress if they were sudden and unexpected. The following quotation from a patient's account of his symptoms illustrates this. "At these times I noticed another feature, my reaction to loud noises such as children's voices, noisy planes, road traffic, my landlady clearing my table after meals and loud music. I can only describe it as if my head were on wires." For this reason the hearing on the affected side may be a definite handicap and if, as is usual in advanced cases, it is severely impaired, there is nothing to be gained, in cases in which only one side is affected, in trying to save it. The tinnitus commonly fluctuated with the deafness. It was by no means unusual to find two types of tinnitus in the same case, a constant hum or buzz with the addition before and during an attack of a jangling machinery-like noise.

A genuine loss of consciousness during an attack must be extremely rare and was only noted in two instances in the present series.

The turbulent eye movements that accompanied a severe attack often caused a temporary impairment of vision though no instance of genuine diplopia or loss of vision without loss of consciousness was ever noted.

The increased activity of the stomach and biliary apparatus combined with the constant retching sometimes resulted in considerable pain in the upper part of the abdomen, and three patients had been actually submitted to abdominal section before the true

nature of the disturbance was suspected. In this connection it has been found that this pain, combined with other manifestations of a vagal disturbance, has at times diverted attention towards the heart and the possibility of coronary heart disease has been considered. Activity may spread to the lower intestinal tract and the resulting spontaneous evacuation of the bowel serves to increase the misery of the sufferer.

Some form of discomfort in, or more often behind, the affected ear was often described. It might be a feeling of numbness, of pressure, or tension, or sometimes there was pain. Very occasionally a definite hemicrania was associated with the attacks, which no doubt accounts for the use of the term vestibular migraine.

Spontaneous nystagmus was only seen during and for a short while after an attack. It might be expected that the disturbance of balance accompanying an attack would end when the nystagmus disappeared. In some patients, however, the feeling of insecurity engendered by repeated attacks so wrecked their self-confidence that they were unable to carry out a simple test of balance long after an attack had worn off.

The caloric test which we have employed during the past five years for the investigation of the vestibular apparatus has been used in all these cases and has rarely failed to reveal a lack of balance between the two labyrinths, the commonest finding being a depression of function on the side of the deafness. Details of this test[14] and its application to Ménière's disease have already been published.[8, 19] It has been found that this test yielded valuable information on the state of the vestibular apparatus in cases of Ménière's disease which enabled an estimate of the location and extent of the vestibular damage to be made. It affords almost the only, and in our experience certainly the most consistent, physical sign of a vestibular disorder, and as each labyrinth is tested separately, it is possible to say with a reasonable degree of certainty whether or no the disorder is limited to one labyrinth. This point is of paramount importance if the possibility of surgical treatment is under consideration. In a previous communication,[8] it was suggested that, on the basis of the cochlear findings, the disease was commonly bilateral though the vestibular findings usually showed changes in one labyrinth only, nearly always but not invariably on the same side as the deafer ear. In the present series of cases the caloric test revealed definite bilateral vestibular involvement in 47 cases (approximately 10 per cent) and these findings did of course influence the decision as to the best type of treatment to adopt.

The sudden translation from normal health to helpless misery, sometimes without warning, is sufficient to unnerve the strongest character. Most patients and not a few of their medical attendants find it difficult at first to believe that such a profound and widespread disturbance is caused by such a modest organ as the labyrinth. Therefore the sense of insecurity engendered by the sudden attacks combined with the fear that some vital process may be in jeopardy can reduce a sufferer to a state of apprehension almost bordering on terror. To quote again from an intelligent patient of even temperament, "what was demoralizing still more was when I got these attacks when out to tea with friends and on more than one occasion I had to be brought home by ambulance. In the end I had to give up all visits—it was nerve racking." The psychosomatic aspect of Ménière's disease is stressed because the disproportion between the symptoms and the signs that can be elicited in a general overhaul combined with signs of worry or mental stress makes, at times, the diagnosis of a functional nervous disorder very tempting.

Diagnosis. In established cases the diagnosis was rarely in doubt. The paroxysms of vertigo alternating with intervals of freedom, the perceptive deafness, almost invariably more noticeable in one ear, and the absence of any signs of disorder within the central nervous system presented a characteristic picture that was seen in all the cases in this series.

The most significant feature was the contrast between the prostrating attack and the intervals—often of weeks or longer—of complete freedom from symptoms of vestibular disturbance.

Conditions of the central nervous system in patients who were seen on account of vertigo included tumours of the eighth nerve, disseminated sclerosis, tumours involving the brain stem, epilepsy and intracranial vascular disturbances. In these cases, however, the pattern of the vertigo and the presence of stigmata specific to each disease formed the basis for differentiation.

Disorders of the vestibular apparatus other than those due solely to distention of the endolymphatic system were often found to resemble closely that condition. All patients in whom an attack was induced by sudden head movements, in whom the condition dated from a head injury, in whom there was a history of past or present otitis media, or in whom an inflammatory process within the labyrinth was suspected, were excluded from the present series. Whilst it is not inconceivable that some of these conditions may have the same pathological foundation as Ménière's disease, it was felt that

further evidence of such a connection was needed before their inclusion as part of the Ménière group would be justifiable.

Many cases in which momentary dizziness was associated with cardio-vascular disease were seen. The trifling disturbance caused by these attacks, usually precipitated by movement, the absence of any signs of vestibular disorder combined with evidence of cardio-vascular disease clearly indicated the cause of the dizziness. Vaso-vagal attacks and other forms of syncope were also seen, but the history, the nature of the attack, and in particular the loss of consciousness in the absence of any aural signs enabled the labyrinth to be excluded.

No case of vertigo due to a purely ocular disturbance has been seen. Several patients with ocular nystagmus have been examined, but in all it has been possible to exclude a labyrinthine cause.

Some disturbance of balance is frequently complained of in certain forms of functional nervous disorders, particularly in anxiety neurosis. The uncomplicated neurotic rarely has an hallucination of movement, and a common story is one in which things appear to fade away or in which there is a fear of overbalancing. If, however, the history is consistent with Ménière's disease, the presence of psychological symptoms, no matter how gross, should never prevent a careful examination of the labyrinth in case an underlying cause for the whole trouble may be found therein. Many patients exhibiting obvious signs of neurosis have been found to be suffering in addition from Ménière's disease.

Treatment. Once the diagnosis has been established the first step in the treatment of Ménière's disease is to ensure that the patient appreciates the underlying cause of the condition. If the explanation offered is acceptable—and in a high proportion of patients it is thankfully received—it does away with the fears and doubts that may have been fostered or even engendered by previous professional or friendly counsel.

It has been found that the proportion of cases in which the attacks cannot be abolished or controlled sufficiently to enable a patient to lead a life of useful activity is very small indeed. Cases in which the vestibular end-organ on both sides is actively affected may prove very stubborn and account for nearly all the failures to relieve the condition.

Nevertheless, it is very necessary that the patient should, at the very outset, be made to realize that although the attacks can

usually be abolished or controlled, this does not necessarily mean the entire disappearance of all symptoms. Generally speaking, conservative treatment tends to smother the attacks rather than abolish them, whilst surgical interference, though it prevents further attacks, leaves some residual dizziness, particularly provoked by sudden head movements, and also a tendency to tire easily after physical exertion.

Many different forms of treatment have been advocated and attention is drawn to a comprehensive review of the disease by Harrison[23] in which all the current forms of treatment are discussed. The views that I have previously expressed on the treatment of Ménière's disease[5] are still the same in the light of further experience, so I will confine myself to some general observations on the management of these cases, together with results to date of the operative procedure originally described in 1943.[5]

All the cases under review have been given a trial with conservative treatment, and in the majority this has been sufficient to control the attacks. By the time that they reach me, most patients have already been started on a course of phenobarbitone. This popular sedative does undoubtedly seem to discourage the spread of the disturbance caused by an attack to other parts of the central nervous system. In patients who can stand it, small doses of hyoscine—the basis of many seasick remedies—have sometimes been of great help in modifying the attacks.

It has, however, been found that the exclusion of salt and salty foods from the diet, combined with a limitation of the fluid intake, has resulted in a greater measure of relief than any other form of conservative treatment. Of recent times it has in Great Britain been extremely difficult to adhere to any form of diet, but the following regime, modified from a diet kindly suggested by Dr. R. A. McCance, can even nowadays usually be followed without undue hardship.

LOW-SALT AND RESTRICTED FLUID DIET

1. Neither salt nor soda is to be used in the cooking or at the table.

2. The following foods MUST be avoided:—

Bacon	Tinned Fish
Ham	Bloaters
Corned Beef	Kippers
Salt Beef	Smoked Fish
Sausage Meat	Shell Fish
Meat Extracts	Baked Beans
Gravy	Breakfast Cereals
Tinned Soups	Bottled Olives
Marmite	Meat and Fish Pastes

3. Cheese and salt butter should be taken sparingly if they cannot be avoided.

4. Bread should be baked free of salt when practicable.

5. Not more than 2½ pints of fluid should be taken in any 24 hours.

In some cases, in addition, injections of histamine have been given in accordance with the method described by Shelden and Horton[31] sometimes with considerable benefit. In view of Wright's[33] findings, a careful search has always been made for an obvious focus of sepsis in the nose, mouth or throat, and whenever one has been found steps have been taken to see that it is eliminated. The number of cases has not, however, been sufficiently large to support the view that infection in the nose or throat plays an important part in the causation of Ménière's disease.

Patients whose attacks were not controlled by conservative treatment, in whom only one vestibular labyrinth was affected, and in whom the hearing on the same side was obviously impaired, were considered suitable cases for destruction of the end-organ. This was usually effected by removing a piece of the membranous external semicircular canal, though it has been found that the same result followed merely tearing it across. This procedure, as will be seen from the following table, has been employed in 116 cases without any untoward result.

Fig. 9.—Membranous external semicircular canal removed at operation. It has been stained with alum carmine and mounted in glycerine jelly. (x10)

SUMMARY OF OPERATIVE TREATMENT

Saccus Endolymphaticus incised		5
Labyrinthotomy		130
	Alcohol injected	10
	Membranous canal diathermy	4
	Membranous canal excised (Labyrinthectomy)	116
Total Operations		135

Fredom from troublesome complications was attributed to the simplicity of the procedure in terms of general surgical principles. That is to say, a portion of the diseased organ was excised and, except for two cases in which mild sepsis supervened, nothing was introduced that might have a harmful effect on the labyrinth or surrounding structures. The fact that so few complications were encountered in this series of operations is attributed to the fact that in most cases powerful agents such as alcohol and diathermy, over which exact control cannot always be exercised, were avoided.

Under general anesthesia and through a postauricular incision the external semicircular canal is exposed via the mastoid, and the incus is removed to give better access to the canal.

Using a Leitz binocular dissecting microscope with a working distance of 22 cm. and giving a magnification of 10 diameters, the bony external semicircular canal is opened with a dental drill using a diamond paste burr. The membranous canal is identified and removed with fine ophthalmic forceps, after which the mastoid wound is closed without drainage. The operation takes from one-half to one hour, and apart from two instances of mild sepsis has not been followed by any complication.

IMMEDIATE RESULTS OF OPERATIVE TREATMENT.

Total Operations	135
Healed by first intention	133
Low-grade infection with ear discharge for one week	2
Other complications	0
Deaths	0

LATE RESULTS OF OPERATIVE TREATMENT

Total Operations	135
Cochlear and vestibular function abolished	133
Not abolished (two saccus cases)	2
General state and ability to work	
Improved and able to work	120
Not improved and unable to work	9
No record	6

Following this operation there has, save in two saccus cases, been a complete loss of both cochlear and vestibular function. In this connection it may be mentioned that one of the principal objections to this or any other form of operation on the labyrinth which results in loss of cochlear function has been that potentially useful hearing has been sacrificed. It must, however, be pointed out that in nearly all the cases operated upon the hearing on the affected side was not only seriously impaired, but, in virtue of the nature of the lesion causing the impairment, it was also so distorted as to be a handicap

rather than a potential asset. Therefore I think that it is fair to say that unless there is good reason to suppose that both ears are actively involved, no useful object can be served by trying to preserve a distorted remnant of hearing.

A particularly interesting feature of the immediate post-operative period has been the absence of any ocular signs of labyrinthine disturbance whilst the patient was still unconscious. In no case has any deviation of the eyes been noted, and it has not been until the patient was fully conscious that nystagmus was seen. Once conscious, however, the symptoms and signs of acute vestibular failure were evident. It has been found that the degree of postoperative disturbance is in direct proportion to the amount of preoperative vestibular function and in any case is likely to be considerable. For this reason it has always been found wise to warn the patient beforehand that for a few days after operation there may be an exacerbation of the symptoms from which relief was sought.

A programme of rehabilitation which includes a series of special head exercises is always employed for these as well as for other cases of vestibular injury,[6] and whenever practicable this is instituted before operation. Most patients are able to leave their bed within a week and the hospital within three weeks. Some residual dizziness, particularly that provoked by sudden head movements, is to be expected, and sustained physical exertion may often result in undue fatigue. With but very few exceptions patients are glad to return to work within two months of operation.

The opportunity of seeing and treating these cases has been made possible by the kindness of my colleagues at King's College Hospital, the National Hospital Queen Square, Horton Emergency Hospital, and of friends elsewhere who have referred cases to me. I am particularly indebted to my friend and colleague Dr. C. S. Hallpike for his constant cooperation and advice and also to Dr. A. B. Hewlett for his help. I should also like to record my thanks to my assistants for their help, and in particular to Miss E. M. Whetnall and Mr. P. H. Jobson for their aid in collecting together the material for this paper.

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III

ANTIBIOTICS IN THE TREATMENT OF SINUS INFECTIONS

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During a single decade the indications for sinus surgery have become greatly modified because widespread clinical trial has demonstrated the ability of the sulfonamides, and later of the antibiotics, to alter profoundly the course of many acute and some chronic infections. The change in criteria for radical intervention is as significant as the fact that the new agents promptly control many acute sinus infections. The majority of acute sinus infections were adequately managed before the introduction of chemotherapy. The main value of the antibiotics and the sulfonamides lies in their ability to control infections resistant to ordinary rhinologic procedures so that surgical measures are either avoided or limited to more simple procedures.

Of the many known antibiotic substances only penicillin, streptomycin and tyrothricin have attained a position of therapeutic importance but to each of these a specific therapeutic sphere can be assigned on the basis of our present knowledge. The antibiotics cannot be regarded as entirely displacing the sulfonamides. In the treatment of some infections the sulfonamides and the antibiotics supplement each other. In the treatment of other infections the drugs of one or the other group may be used to replace each other when the infecting microorganisms develop resistant strains or the patient acquires sensitivity to the drug. The economy and plentiful supply of the sulfonamides are advantages often outweighed by the comparative safety and absence of toxicity of penicillin, especially in extremely ill or debilitated patients and in the presence of liver and kidney damage.

A definite reduction in the number of sinus operations has been effected since the introduction of the new therapeutic agents. Since it is unlikely that the indications for surgical intervention in the

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Read at the meeting of the Eastern Section of American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 13, 1947.

presence of neoplasms or tissue alterations induced by allergy have been appreciably affected by the introduction of chemotherapeutic agents, it is reasonable to assume that more infrequent surgery has been employed in the management of acute and chronic infections and of certain traumatic fractures of the paranasal sinuses.

The antibiotics have proven to be of little or no value in uncomplicated allergy but they have been effectively used against secondary infections complicating allergy of the upper and lower respiratory tracts. Thus, there is reason to expect that bacterial sensitization may sometimes be forestalled in those instances where infection in an allergic individual can be arrested or eradicated by antibiotic therapy. The prophylactic use of the antibiotics merely to prevent secondary infection cannot as yet be justified because allergic patients are prone to become sensitive to the antibiotic drugs themselves, and this is especially apt to occur when the drug is administered as a prophylactic measure over a long period of time.

Sinus surgery continues to be indicated in the presence of advanced, irreversible tissue change in the nasal passages and sinus cavities even when attributable to allergic influence alone, and for the reasons generally accepted prior to the introduction of the antibiotics. When secondary infection of allergic tissues takes place, it is less amenable to chemotherapy than is similar infection in otherwise normal tissues. Chronic infection in sinuses containing extensive, irreversible tissue changes due to allergy can rarely be influenced by chemotherapy alone and it eventually demands some form of surgical attack.

The generally accepted rule that as little surgical intervention as possible should be employed in acute sinus infection has become even more rigid since the introduction of chemotherapy. Exceptions to this rule were formerly conceded in the presence of orbital extension of the sinus infection, bacteremia, or retrograde thrombophlebitis. Timely chemotherapy has eliminated most of these exceptions. Thus penicillin will rapidly control orbital cellulitis of sinus origin, clear the blood stream of viable organisms and impede the progress of intravenous extension of the infection. Once appreciable collections of pus exist, however, drainage must be established in spite of continuous chemotherapy.

With or without chemotherapy, most acute sinus infections should be permitted to become chronic before embarking upon surgical attack. Such delay is justified by the fact that properly directed chemotherapy in recalcitrant acute infections of the sinuses is often

capable of forestalling the chronic types of infection that lead to surgery.

The administration of antibiotics in mild and transient sinus infections must be regarded as poor therapeutic practice. It has long been recognized that immunity often fails to be acquired when an infection is promptly controlled by chemotherapy. The prompt arrest of bacterial activity and of the tissue reactions incident to bacterial invasion prevents positive stimulation of immunological responses. As a result, there are frequent recurrences at very short intervals. The tendency to readminister the chemotherapeutic agent at each recurrence seems justified by the success attending its use during the first attack. Under these circumstances, however, repetition of the courses of chemotherapy fails to prevent progressive tissue changes, fosters the development of resistant strains of microorganisms, and may sensitize the patient to the drug. In the latter event, the drug is then disqualified for the treatment of a subsequent and perhaps more serious infection. It is when prolonged, continuous chemotherapy has rendered the pathogen resistant to the drug that infection may spread to previously uninvolved sinuses during the active course of treatment with antibiotics. Because of the interference of chemotherapeutic agents with immunological responses, the concomitant use of autogenous vaccines or lysates deserves serious consideration as a means of avoiding recurrences.

Antibiotic therapy is not a cure-all. It is effective only when directed against sensitive microorganisms. Whenever possible, sensitivity tests should determine the vulnerability of the pathogen before treatment is started. In the presence of resistant strains, antibiotic therapy is doomed to failure. Meanwhile, many time-tried therapeutic measures are neglected to the ultimate detriment of the patient.

Chronic sinus infections are, in the main, resistant to antibiotic therapy because the drug penetrates with great difficulty those abnormal tissues resulting from repeated or prolonged infection. Tyrothricin lacks the ability to penetrate tissue and its toxicity in other than surface use renders its value in the treatment of chronic sinus infections highly questionable. Even though penicillin and streptomycin control the chronic infection temporarily, recurrence frequently follows withdrawal of the drug. The action of penicillin is not inhibited by organic matter as is the action of the sulfonamides, but the drug is physically unable to penetrate accumulations of pus containing susceptible and viable pathogens. Under such circum-

stances surgical drainage is still indicated. Streptomycin actually loses some of its antibacterial property in the presence of body fluids. Thus the tolerance of gram-positive cocci to streptomycin is increased four to eight times in body fluids. If the clinical and laboratory findings warrant it, both penicillin and streptomycin may be used in resistant cases. The toxic properties of streptomycin are less marked than those of the sulfonamides but greater than those of penicillin. Among the more serious effects of streptomycin are involvement of both the vestibular and cochlear fibers of the eighth nerve, allergic-like reactions in the skin and joints, and certain kidney effects. While streptomycin must be used against penicillin-fast microorganisms, penicillin is the antibiotic of choice wherever its effectiveness is indicated.

Because eradication of chronic infections in the sinuses cannot always be accomplished by surgery alone, antibiotic therapy as an adjunct to surgery may play a decisive role. The combined local and intramuscular use of penicillin, after surgical failure, frequently is followed by the disappearance of exudate and viable organisms. Thus far, the most satisfactory results in the management of chronic sinus infections, with advanced irreversible tissue changes, have been obtained by the combined use of surgery and chemotherapy.

In infectious sinus diseases, effective antibiotic therapy during the preoperative period depends upon early and accurate diagnosis (especially bacteriological diagnosis), followed by selection of the antibiotic most lethal to the invading organisms, given in adequate dosage by the most effective route of administration, and over a sufficient period. Because antibiotic drugs can impede or arrest the spread of infection, minimize the occurrence of bacteremia and the toxic effects of an active and overwhelming infection, there is an appreciable prolongation of the "interval of safety" between the onset of symptoms and the need for surgical intervention. This delay affords an opportunity for detailed studies prior to operation and meanwhile recovery may be accomplished without surgery.

A sense of false security may be engendered by the prompt therapeutic effects of the sulfonamides or the antibiotics in those sinus infections in which irreversible tissue changes have taken place. Recurrence is the rule when chemotherapy is withdrawn and although the recurrent attacks may appear to subside with resumption of chemotherapy, complete control of the infection is not obtained until surgical measures are employed.

During the period of operation, antibiotics, used locally and systemically, have prophylactic value in the prevention of meningitis, the control of bacteremia and the protection of uninfected tissues in the operative field from contamination.

Throughout the postoperative period, the antibiotics limit the spread of infection to freshly exposed tissues and tend to shorten the healing period.

Fractures of the paranasal sinuses with a tear of the overlying dura were almost invariably followed by meningitis. The prompt administration of antibiotics has materially reduced the incidence of this complication. Where meningitis has already followed such an injury, antibiotic therapy will often bring about recovery from the meningitis before any attempt is made to repair the dural defect. In fact, under these conditions, better clinical results have been obtained when dural repair has been delayed until recovery from meningitis has occurred.

Under continuing antibiotic therapy, many fractures of the paranasal sinuses heal by primary union. Bone fragments, if surrounded entirely or even in part by vascular soft tissue, exhibit amazing viability, but those lying free within a sinus cavity require eventual removal. The frequency of sequestrum formation and subsequent surgical removal has been greatly reduced since the introduction of the antibiotics.

The ears and the paranasal sinuses are the most frequent sites of primary foci in meningitis. In the past, surgical drainage of the primary focus has been axiomatic in the treatment of meningitis. Clinical trial has demonstrated that an initial attack of meningitis can be controlled by chemotherapy with or without surgical drainage. The combined use of an antibiotic and a sulfonamide has generally proved to be more effective than a single drug of either group and this is especially true when the pathogen is a pneumococcus. Thus considerable doubt has been expressed concerning the value of drainage of primary foci when chemotherapy is used. This view is not generally accepted. A combination of antibiotic and sulfonamide therapy plus eradication of the primary focus appears to give the most consistent permanent result.

Even though chemotherapy may not eliminate the need for surgical drainage, its delaying action is of great value. Surgical intervention is delayed until the meningitis is controlled. The primary focus can then be attacked when the results of precision laboratory

and roentgenological studies are at hand and the patient has become a better surgical risk.

It has been a common experience to encounter relapsing attacks of meningitis more frequently in sinus infections than in mastoid infections. This alone emphasizes the importance of surgical drainage of a primary focus located in the sinuses. Certainly drainage cannot be safely avoided in the treatment of the relapsing case even though its value may be regarded as questionable during the acute phase of meningitis. Occasionally the full recovery of a patient with relapsing meningitis can be attained with chemotherapy alone, but usually a chronic focus consisting of a formidable collection of pus and sequestra will not remain quiescent indefinitely. Eventually such a focus must be dealt with surgically and especially when reinfection of the meninges coincides with discontinuation of the chemotherapy.

When in the attempt to prevent relapse, surgical drainage of an extradural source of infection produces disappointing results, the failure should not be ascribed to the futility of surgical intervention but rather to incomplete surgical drainage of a single focus or the presence of multiple foci which escape recognition. In the presence of an extensive primary focus, surgical intervention cannot be safely disregarded, and the concurrent use of chemotherapy and surgical drainage should be the method of choice.

Frank osteitis accompanying acute sinus infections is promptly controlled by adequate doses of penicillin, but occasionally the withdrawal of penicillin is followed by reactivation of the infection and extension of the infection to the meninges. The meningeal infection usually responds to further penicillin administration thus permitting delay in operation. Surgical intervention has been more often successful when carried out after control of the meningitis.

A feared complication of frontal sinus infections is osteomyelitis. With or without brain abscess, osteomyelitis of the frontal bone has rarely been cured by antibiotic therapy alone. Surgical intervention with maintenance of adequate drainage continues to be imperative. The record of surgery alone is not too brilliant but the concurrent administration of penicillin, intrathecally and intramuscularly, together with surgical drainage has effected some remarkable recoveries. In spite of the virtues of antibiotic therapy in controlling osteomyelitis of the frontal bone, all necrotic bone must be removed before healing can occur. While the antibiotics exhibit a peculiar ability to relieve the pain associated with osteomye-

litis, their main value lies in the prevention of sequestration and in the arrest of progressive extension to contiguous osseous tissue. The timely use of antibiotics in osteomyelitis secondary to sinus infection has greatly reduced the extent of radical surgery but has not eliminated the need for surgical drainage.

CONCLUSIONS

1. Chronic sinus infections are less responsive to antibiotic therapy than are acute sinus infections.

2. In the presence of pus or of sequestra, the use of antibiotics fails to eliminate the need for surgical intervention.

3. The use of antibiotics in the treatment of sinus infections has resulted in a significant reduction in the incidence and extent of sinus surgery.

1912 SPRUCE STREET.

IV

THE HISTOLOGIC EFFECT OF REPEATED APPLICATION OF CERTAIN NOSE DROPS TO THE NASAL MUCOUS MEMBRANE OF RABBITS

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ROCHESTER, MINN.

The excessive use of nose drops for a prolonged time by human beings has been observed to have caused a clinical syndrome which may be designated "vasomotor rhinitis medicamentosa." The patient who subjects his nasal passages to overmedication usually does so to relieve a sensation of congestion. The congestion may be due to normal physiologic activity of the nasal membranes or to certain pathologic conditions of the nasal membranes. The relief afforded becomes less marked as the practice is continued; finally, little or no relief is obtained. Advice is then sought from a rhinologist.

The use of nose drops may have been initiated on the patient's own responsibility because of the persuasive effect of advertising by the manufacturers or in some instances on the advice of physicians not thoroughly familiar with the physiology of the nose.

The most effective treatment for the relief of the syndrome has been found to be discontinuance of the use of the nose drops.

A study was undertaken to observe the histologic changes in the nasal mucous membranes produced by the overuse of nose drops. Rabbits were used in these experiments. The rabbits were kept under the same living conditions in the same room, fed the same food, and cared for in the same manner.

A normal rabbit was kept for a period of three weeks as a control under the same conditions as the other rabbits. A specimen of the nasal membrane of this rabbit was removed and studied histologically. The histologic sections of this specimen have been used as a normal for comparison (Fig. 1).

Abridgment of thesis submitted by Dr. Ryan to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of M.S. in Otolaryngology and Rhinology.

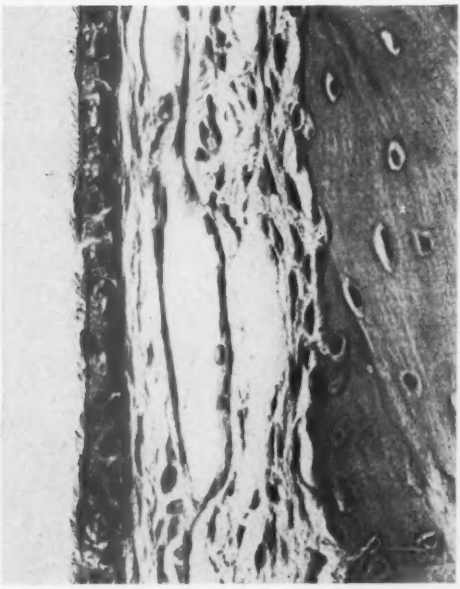
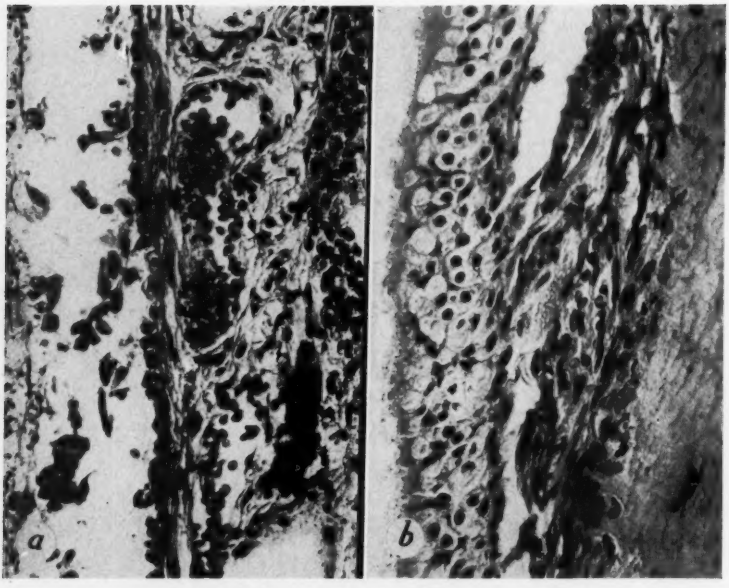


Fig. 1.—Normal nasal mucous membrane of a rabbit that was used as a control (x430). The ciliated columnar surface epithelium is clearly evident. Under the surface is the normal subepithelial tissue containing blood vessels of normal size and contour. There is a complete absence of inflammatory cells. The density and the depth of the subepithelial structure are very marked.

Nose drops were applied to the nasal membranes of each of the other rabbits in the same manner. The rabbits were restrained and the medicine was administered by the dropper method four times each day. At various intervals of time, specimens of mucous membrane were removed, fixed, embedded in paraffin, sectioned and stained, and examined microscopically. These histologic sections were compared with the sections obtained from the normal rabbit. The changes which occurred in the tissue during the time the drops were applied were then compared.

Two series of rabbits were used. Two types of commercial nose drops were employed. To facilitate description, these will be designated as "Preparation 1" and "Preparation 2." Preparation 1 contained 0.125% of dl-desoxyephedrine hydrochloride in a stabilized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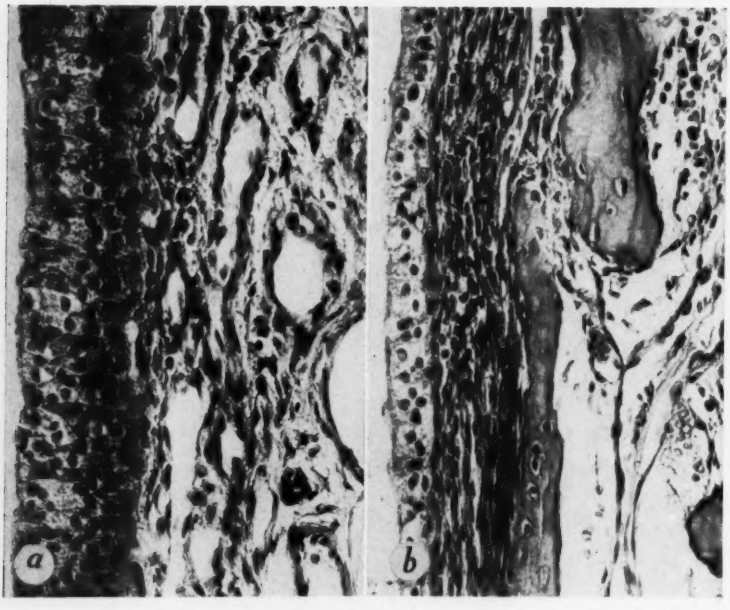
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Fig. 2.—*a*. Nasal mucous membrane of a rabbit that had received applications of Preparation 1 for six days (x300). The blood vessels are greatly dilated. There is a surface exudate which is mainly mucous in character. The cilia are completely absent and the superficial epithelial cells have been destroyed. There are some subepithelial edema, a suggestion of beginning edema of the epithelial cells and many inflammatory cells which consist chiefly of lymphocytes and plasma cells. *b*. Nasal mucous membrane of a rabbit that had received applications of Preparation 1 for 11 days (x300). The epithelial cells show an extreme degree of edema. The surface cells of the epithelium show signs of degeneration. The blood vessels are greatly dilated.

Fig. 3.—*a* Nasal mucous membrane of a rabbit that had received applications of Preparation 1 for 35 days (x300). No cilia are present. The epithelial cells and subepithelial connective tissue show evidence of edema and degeneration. The dilatation of the blood vessels is not as great as that shown in Fig. 2. The inflammatory cells also are fewer in number. *b*. Nasal mucous membrane of a rabbit that had received applications of Preparation 1 for 44 days (x300). No cilia are present. The epithelial cells show evidence of a considerable degree of edema and degeneration. The subepithelial layer is dense, fibrinous and avascular. Only a few inflammatory cells are present. Some edema is present in the subepithelial layer.

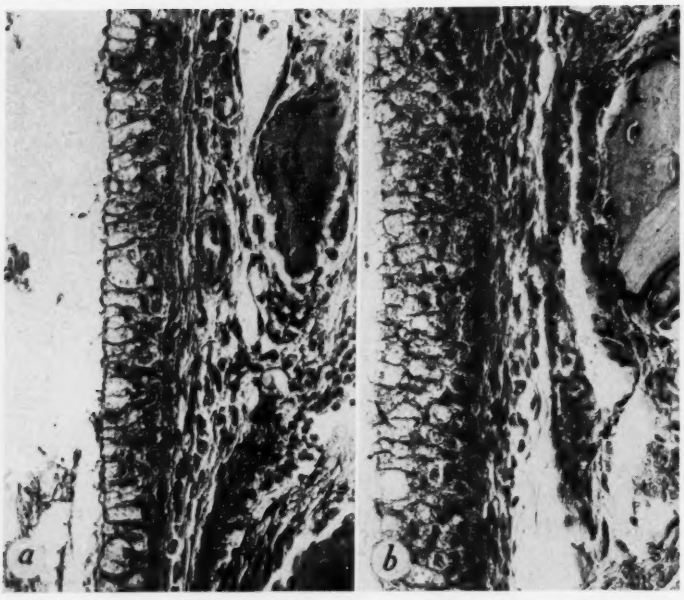


Fig. 4.—*a*. Nasal mucous membrane of a rabbit that had received applications of Preparation 1 for 60 days (x300). A mucocellular surface exudate is present, but cilia are absent. The epithelial cells are edematous, are filled with mucus and have the appearance of goblet cells. The blood vessels are constricted. The subepithelial layer of tissue is much denser than normal and edema is present in the cells of this layer. There is an absence of inflammatory cells. *b*. Nasal mucous membrane of a rabbit that had received applications of Preparation 1 for 70 days (x300). There is a complete absence of cilia. The epithelial cells show edema and degenerative changes. There is a tendency toward stratified squamous cell metaplasia in the epithelial layer. The absence of inflammatory cells is noteworthy. The blood vessels are constricted and there are signs of edema in the structures of the subepithelial layer.

aqueous vehicle, 1.25% of anhydrous sodium sulfathiazole and 1.25% of sodium sulfadiazine. The sulfathiazole and sulfadiazine had been added to inhibit the multiplication of secondary pathogens which cause complications in cases of coryza and nasal infection. The manufacturers of Preparation 1 have warned that it should not be used by persons who have exhibited a sensitivity to the sulfonamide drugs. Preparation 1 was used because it contained ephedrine, which is a common constituent of nose drops.

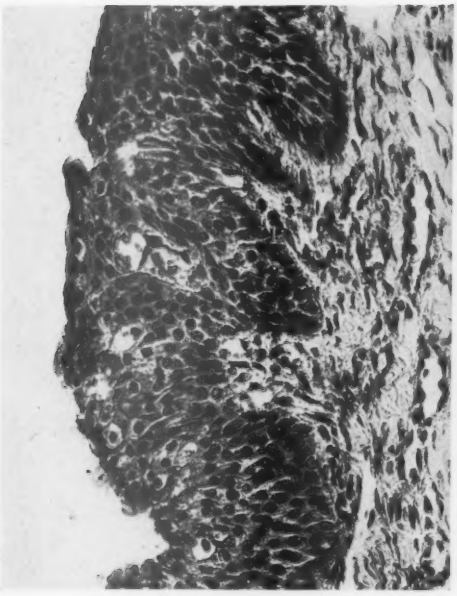
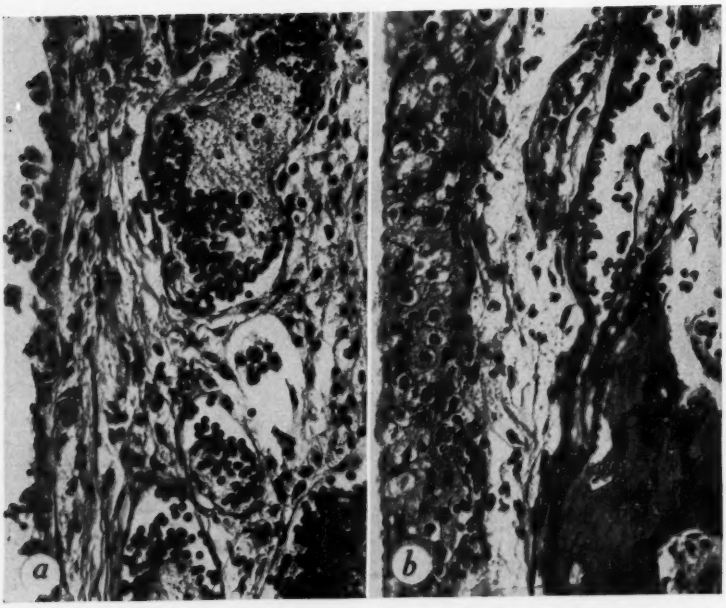


Fig. 5.—Nasal mucous membrane of a rabbit that had received applications of Preparation 1 for 80 days (x300). The cilia are absent and the epithelium is of the stratified squamous cell type. The blood vessels are constricted. The subepithelial layer is thickened and some fibrosis is present.

Preparation 2 was a solution of 2-naphthylmethylimidazoline hydrochloride, which is a member of the organic compounds characterized by the imidazoline ring. Preparation 2 was selected for the following reasons: (1) at the time of this study, it appeared that the general public was using this preparation as frequently, if not more frequently, than any other type of nose drops and (2) the preparation appeared to be causing a strong postcongestion reaction, as determined by clinical examination. It should be stated, however, that practically all types of nose drops produce this reaction.

This drug acts on the peripheral vascular bed by causing vasoconstriction. The site of its action is the peripheral sympathetic nerve endings. In this respect, it resembles epinephrine. Pharmacologic observations have shown that the resulting vasoconstriction of the peripheral vessels is of the same degree as that induced by epinephrine but that it persists for a much longer time. The solution for nasal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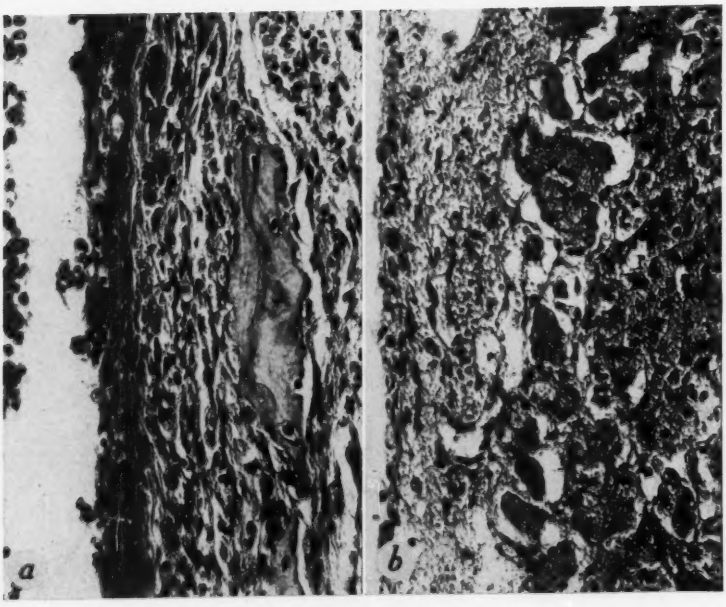
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Fig. 6.—*a.* Nasal mucous membrane of a rabbit that had received applications of Preparation 2 for eight days. The blood vessels are greatly dilated. There is a minor degree of inflammation. The cilia have been destroyed and there is definite evidence that the epithelium has undergone extensive destruction. There is some evidence of tissue edema. *b.* Nasal mucous membrane of a rabbit that had received applications of Preparation 2 for 17 days (x300). There is a moderate amount of both epithelial and subepithelial edema. Considerable dilatation of the blood vessels is present. The cilia have been destroyed completely and epithelial destruction is present. Inflammatory cells may be seen throughout the subepithelial tissue.

Fig. 7.—*a.* Nasal mucous membrane of a rabbit that had received applications of Preparation 2 for 32 days (x300). A cellular surface exudate is present. Complete absence of cilia and destruction of the surface epithelium can be seen. Some suggestion of an attempt toward a change to stratified squamous cell metaplasia is evident. Inflammatory cell infiltration of the polymorphonuclear type and spindle cell proliferation of connective tissue can be ceen. *b.* Nasal mucous membrane of a rabbit that had received applications of Preparation 2 for 50 days. A considerable amount of subepithelial fibrosis and an increase in the subepithelial connective tissue layer can be seen. The blood vessels are not dilated. The cilia are completely absent. No evidence of a normal pattern of the surface epithelium is present. Inflammatory cells are not an important feature at this stage.

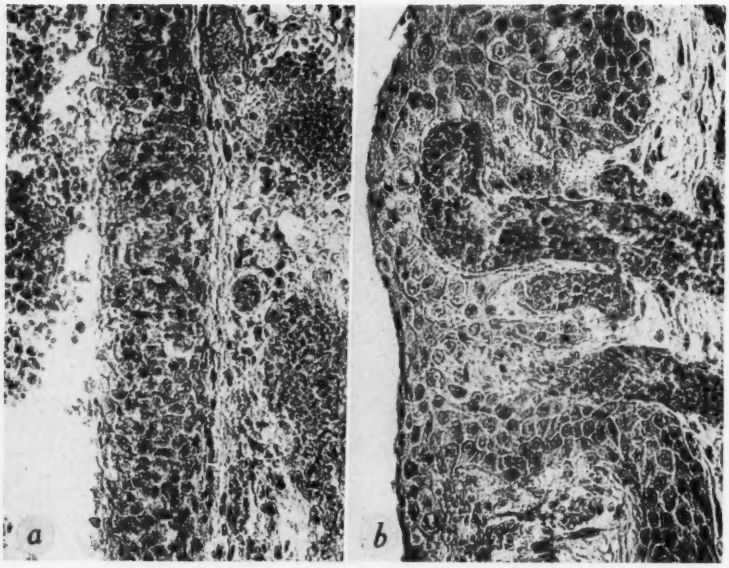

Fig. 8.—*a*. Nasal mucous membrane of a rabbit that had received applications of Preparation 2 for 60 days (x300). The surface epithelium shows stratified squamous cell metaplasia. The cilia are completely absent. A cellular surface exudate is present. An occasional erythrocyte is seen in the subepithelial layer. Very few inflammatory cells are present. *b*. Nasal mucous membrane of a rabbit that had received applications of Preparation 2 for 70 days (x300). There is a complete conversion of the surface epithelium to the stratified squamous cell type by the process of metaplasia. Proliferation of blood vessels is present and the blood vessels seem to be infiltrating the squamous epithelium. Cilia are absent completely. There is no evidence of the presence of inflammatory cells.

medication is an isotonic aqueous solution with a pH of 6.2. It, therefore, is slightly acid. It has been claimed that this solution is not injurious to the nasal mucous membrane, that it does not produce undesirable local and systemic side effects, that it has a pH which approximates that of clinically normal nasal secretions, and that it does not interfere with ciliary action. The medium in which 2-naphthylmethylimidazoline hydrochloride is dissolved contains a balanced amount of phosphates, chlorides and dextrose to preserve isotonicity and the slightly acid reaction. It is this medium which

is claimed to constitute the buffer system to correct the pathologic alkaline pH of an inflamed mucosa and to induce the normal, slightly acid reaction and to maintain this corrected pH for long periods. The solution is available in two strengths: a 0.1% solution and a 0.05% solution. The stronger solution is recommended for use by adults only and the weaker of the solutions is advocated for children and for persons with an extremely sensitive nasal mucosa. The 0.1% solution was used in this study.

At the beginning of the study, several young adult rabbits died within a few days after the instillation of the drops was begun. The majority of the rabbits that died had been receiving Preparation 1. This may have been a mere coincidence. The rabbits that died unexpectedly showed no pathologic process, for example, no changes other than slight pulmonary inflammation and congestion.

RESULTS

Application of Preparation 1.—Histologic examinations revealed that cilia were present on the nasal mucous membrane after Preparation 1 had been applied for three days but no cilia were observed in any of the sections obtained after this preparation had been applied for five days (Fig. 2*a*).

Dilatation of the blood vessels of the nasal mucous membrane occurred as early as the third day. This dilatation (Fig. 2*b*) continued until the later stage of the study, when constriction occurred.

Scattered inflammatory cells were observed in the early stage of the study. These cells increased in number until the fourth week and they then gradually disappeared (Fig. 3*a*).

The epithelial cells were damaged as early as the fifth day. During the second week, the epithelial cells showed signs of edema. During the third week, the surface epithelium began to show signs of degenerative changes. These changes persisted through the tenth week. The amount of edema in the epithelial layer of cells (Fig. 3*b*) increased in direct proportion to the length of time the preparation was used. In the latter stages of the study these cells became filled with mucus and resembled goblet cells (Fig. 4*a*); finally, the normal epithelium was replaced completely by stratified squamous epithelium (Figs. 4*b* and 5).

A mucocellular surface exudate was present in all of the sections obtained during the first week and continued to be present into the ninth week.

The subepithelial layer of the nasal mucous membrane showed thickening and fibrosis in the third week; edema was present in the fourth week and remained present throughout the rest of the study.

Application of Preparation 2.—In the cases in which Preparation 2 was applied, the first histologic change that was observed in the nasal mucous membrane was a destruction of the cilia (Fig. 6*a*). This was first observed after the preparation had been applied for eight days and it continued throughout the period of study.

The blood vessels were dilated in the early stages (Fig. 6*b*). After 40 days, the blood vessels became constricted and some even appeared to be sclerosed.

A considerable number of inflammatory cells were present from the earliest stages (Fig. 7*a*). These cells, however, tended to disappear in the later stages of the study and were not noticed in any great number beyond the fiftieth day (Fig. 7*b*).

The epithelial cells became damaged during the first week. On about the seventeenth day of the study, the epithelial cells began to show signs of edema and appeared to be filled with mucus. During the third week, the epithelial cells began to show evidence of degenerative changes and an increase in the edema. During the fifth week, the epithelial layer of cells became very disorganized and bore little or no resemblance to the epithelium of normal mucous membrane. About the seventieth day, a complete change from the ciliated columnar epithelium to nonciliated stratified squamous epithelium occurred (Figs 8*a* and 8*b*).

A mucocellular surface exudate was present during the third week and continued to be present until the study was completed.

In the later stage of the study, the subepithelial layer of tissue became thicker and denser than normal and fibrosis was present. Edema seemed to extend to this layer about the fifth week of the study.

SUMMARY

This paper is based on a study which was made to determine the effect of nose drops on the nasal mucous membrane of rabbits. Two types of proprietary nose drops were used. To facilitate description, the one type has been designated as "Preparation 1" and the other has been designated as "Preparation 2." Each of these preparations quickly destroyed the cilia. There was no evidence of

development of new cilia during the study. The epithelium was transformed into stratified squamous epithelium.

The subepithelial layer was edematous; it became denser and thicker than normal, underwent fibrosis. The blood vessels became dilated and remained dilated for several weeks. Constriction and sclerosis of the blood vessels was observed at a later date. The entire membrane became congested. Inflammatory cells were observed throughout the layers of the membrane during the first week but began to diminish in number in the fifth week. A mucocellular exudate was present on the surface of the mucous membrane.

It may be concluded that the overuse of nose drops is harmful to the nasal mucous membrane of the rabbit. The clinical picture in the rabbits was similar to that observed in human beings who had used nose drops excessively. If nose drops are to be used for the relief of certain symptoms referable to the nose, it would seem important that their use be well advised rather than promiscuous.

MAYO CLINIC.

V

THE TRAINING OF RESIDENTS IN OTOLARYNGOLOGY

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NEW YORK, N. Y.

There is considerable confusion as to what constitutes adequate training for residents in otolaryngology. Because of this confusion, I have attempted to correlate in this paper:

1. The requirements of the American Medical Association for approved residencies.
2. The requirements of the American Board of Otolaryngology for residents' training.
3. To express some thoughts by which residencies in otolaryngology can be made acceptable to these two organizations, as well as to the residents who seek training.
4. To offer a conversion plan to solve these problems which is being set up at Manhattan Eye, Ear and Throat Hospital in New York City.

A residency has been defined as a progressive and graduated educational experience, designed to enable a physician to make himself proficient in a special field of practice and to give him the additional background of continued development in the field. This is in contrast to an internship, which is a part of the basic preparation for the practice of medicine.

Because of the inadequacy of residencies and internships, the terms "Fellow" and "Graduate Student" have been used to designate those pursuing advanced studies in a specialty. A residency as here defined is a complete preparation for a specialty, while a fellowship indicates a greater opportunity for research and the study of the basic sciences. A fellowship is ordinarily a university, rather than a hospital, appointment.

The scope of medical knowledge today is such that to specialize in a particular field requires more preparation in general medicine,

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as well as intensified special study and training, than has ever been required before.

The formation of the American Board of Otolaryngology has stimulated many graduates in medicine to spend another five years in special study.

There are both general and specific requirements for certification by the American Medical Association with reference to residencies in otolaryngology. Under the general requirements, the size of the institution is not so important if the clinical material is such that there are sufficient manifestations for the study of diseases which pertain to this particular specialty. It must be well equipped with appliances and apparatus pertaining to the treatment and diagnosis employed in the specialty in which a residency is offered. It requires that there must be well-qualified and experienced physicians giving instructions to the residents. The Director of a Resident Service should be a competent specialist who is at least eligible for certification by the corresponding Specialty Board. He should assume direct responsibility for the training of residents and should stimulate others of his staff to give instruction and sympathetic cooperation, which residents require. Monthly clinical and pathological conferences, and other regular staff meetings are required to be held, at which clinical observations are reviewed, particularly necropsy reports. In these conferences a resident is given an opportunity to prepare reports, correlate the findings, and train himself to speak before others. The Department should contain roentgenographic and roentgenoscopic as well as radium equipment. The Association requires an adequate medical library, containing modern texts, the "Quarterly Cumulative Index Medicus", and current journals relating to the field of the residency. It requires that the histories must be complete, including a physical examination at the time of admission, preliminary diagnosis, laboratory observations, descriptions of operations, progress, final diagnosis, condition on discharge, end results, and in cases of death, necropsy observations, if obtained. All entries in histories should be signed. The records should be in charge of a trained librarian. Alphabetical and diagnostical indexes of the patients, operative procedures, radiological and pathological material, should be maintained. It specifies that in a combined residency in ophthalmology and otolaryngology, five years of training and experience should constitute the minimum time required. The requirements of the American Medical Association for otolaryngology specifically are as follows:

"The qualifications for special practice in this field include an approved internship and three years of graduate preparation, with completion of an acceptable basic science course and at least one and preferably two years of residency training in otolaryngology.

"Residencies and fellowships in otolaryngology should aim to provide adequate clinical and operative experience and additional opportunities, if possible, for graduate study in the related basic sciences of anatomy, embryology, histology, physiology, pathology, bacteriology and pharmacology.

"Essential equipment for diagnosis and surgery should be available, including an audiometer and other special apparatus depending on the scope of the service. The department should further provide histologic and pathologic material for demonstrations and study and preferably facilities for experimental surgery and dissection of the head and neck. The clinical material should be sufficient in variety and amount to provide adequate training in the various divisions of the service, which might advantageously include the field of bronchoscopy. It is generally assumed that a graduate service would require a minimum of 400 admissions a year and at least 3,500 visits in the outpatient department.

"The training should include a systematic course of instruction with lectures and demonstrations on clinical and technical subjects pertaining to the various phases of otolaryngology. Instruction in surgical technic should be sufficient to enable residents and fellows to undertake operative work on their own responsibility, toward the end of the graduate program."

The requirements of the American Board of Otolaryngology are specifically as follows:

"1. A candidate acceptable for the examination must have been a citizen of the United States (or Dominion of Canada) for at least five years, a graduate of a medical school approved by the Council on Medical Education and Hospitals of the American Medical Association, have had at least one year's general internship in an approved hospital (or its equivalent), and be of good moral character and ethical in his professional relations. Candidates from foreign countries must be permanent residents and citizens thereof, and meet the general requirements above.

"2. A candidate must have membership in the American Medical Association, the Canadian Medical Association, or other medical societies of equal standing recognized by the Council on Medical Education and Hospitals of the American Medical Association.

"3. Each candidate must have had acceptable training in the basic sciences which are fundamental to the intelligent practice of Otolaryngology, including anatomy of the ear, nose, throat, neck, chest, esophagus and related nervous systems, pathology, bacteriology, physiology, didactic otolaryngology and fundamentals of surgery. This requirement may be met by its inclusion in resident training or by a formal postgraduate course.

"4a. *Effective until* 1950: a candidate must have completed a two years' residency in otolaryngology or a three years' residency in Ophthalmo-Otolaryngology and have restricted his practice to Otolaryngology (or Ophthalmo-Otolaryngology) for a minimum of three years or four years respectively, which may include the period of training.

"4b. *Effective after July* 1, 1950: Candidates must pursue a course of three years' study in Otolaryngology, or four years' study if Ophthalmology be included (in which case there must be a minimum of 50% spent in the study of Otolaryngology). The three years may be spent in an approved residency of that duration, which residency may include study of the basic sciences as specified in Section 1; or the candidate may pursue a formal course in the basic sciences, and complete his training with a residency which will give him a total of three years' study in the specialty. In addition to the above, the candidate must have had a minimum of one year in practice limited to the specialty.

"The type of examination is as follows:

"1. Clinical, covering the actual handling of patients, including history-taking, physical and functional examinations, the use of laboratory and x-ray findings and a discussion of differential diagnosis.

"2. Didactic, oral examinations covering all phases of Otolaryngology, including physiology.

"3. Gross and microscopic pathology.

"4. A written examination may be included when the Board deems this advisable."

I may say from experience that "all phases of otolaryngology" may include questions in the fundamentals of surgery, plastic surgery, neck surgery, allergy, certain types of neurosurgery, oral surgery, and broncho-esophagology.

In 1946 a committee was appointed by the American Board of Otolaryngology to draft a suggested program for the training of residents in otolaryngology in the Veterans Administrati. hospitals. This committee, of which I was a member, made the following suggestions, by which a resident might be acceptable for the Board examinations:

"1. The requirements for residencies in V.A. hospitals should be the same as the Board requirements for 1950.

"2. Those hospitals which are operated by the Dean's Committees of recognized medical schools will be considered individually if they have been inspected by the Council on Medical Education of the American Medical Association. The mechanism of such inspections has been set up by the Council on Medical Education.

"3. Residents in these V.A. hospitals should have adequa e training in outpatient or clinic patients and they should also have adequate training in the otolaryngological aspects of pediatric services. Where such outpatients or pediatric cases are not available, the resident should be sent to some other institution where he can obtain such training.

"4. The department of otolaryngology should be independent or autonomous from an operational standpoint. It should have its own instruments and equipment, adequate facilities and equipment for testing hearing and adequate library facilities in textbooks and special journals.

"5. The scope of training should include training in oral surgery, neck surgery, special plastic surgery and some types of neurosurgery."

To this might have been added broncho-esophagology.

In 1945 the American Board of Otolaryngology appointed a special committee, of which I was a member, to evaluate all residencies in otolaryngology in the United States which were approved by the American Medical Association and the American College of Surgeons. Replies from 75 such residencies were received from the following states: California, Colorado, District of Columbia, Connecticut, Georgia, Illinois, Indiana, Iowa, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Missouri, New Jersey, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Tennessee, Texas, Virginia, and Wisconsin. This is a fair sampling. We graded these 75 residencies in otolaryngology as excellent, good, fair, and poor. There were 16 graded as excellent; 27 graded as good; 24

graded as fair; 8 graded as poor. These were graded by the then existing requirements of the American Board of Otolaryngology.

It is evident from a study of these figures that a considerable improvement in resident training is desirable.

It is even more apparent that there is a great amount of work necessary in the conversion of these institutions to comply with the 1950 requirements of the American Board of Otolaryngology. This conversion must start in 1947, in order to train residents for examination after 1950. There is danger that this conversion will not be met unless there is a thorough understanding of its necessity.

Basic Sciences. In order for a resident to get a training in basic sciences at the present time, he must either take a prescribed course at some university which offers such a course, or he must obtain this training in conjunction with his residency. If he is fortunate to have his residency in a hospital which is affiliated with a medical school, he may take his basic sciences on off afternoons or in the evenings.

At the present time, only a few hospitals are affiliated with a teaching university. Residents in a hospital which is not affiliated can not avail themselves of the basic science courses while in residency, unless the hospital itself provides such courses.

There are residents, at the present time, who can not find any opportunity to study the basic sciences.

There is also a great shortage of desirable residencies in otolaryngology. There are comparatively few universities that offer a separate course which can be taken independent of a residency.

The question arises whether a resident receives better training if he takes a certain length of time, namely an academic year, to study his basic sciences or if he takes his basic science training in conjunction with his residency. A year in basic science will also have to be defined as to whether it is an academic year or 12 months. In conjunction with the above statement, one wonders if the student will get as much out of his courses, if he will be too tired after his hospital work to do well in his classes, and if he will neglect his clinical work because of these extra courses. This thought has to be weighed against the definite advantage of being able to apply learning in the basic sciences directly to clinical work at the time the basic sciences are being studied. I am personally inclined to believe that a better understanding in the basic sciences can be attained in conjunction with a residency.

Hard work never hurt anyone, and a hospital offering a basic science course in conjunction with a residency should make it so interesting in its application to clinical material that any fatigue will be quickly forgotten.

Many hospitals already have, or can get, a staff capable of teaching the basic sciences, and I believe that graduate teaching of this type is done much better by those having clinical experience than by those who are trained to teach didactically undergraduate students in medicine.

The undergraduate listens to and accepts didactic teaching, while the graduate student puts the burden of proof on the teacher, especially in the application of his teaching. The graduate student does not accept didactic teaching unless he can apply it.

The American Board of Otolaryngology has prepared a list of basic subjects, the study of which it considers essential in preparation for the examination. They are:

1. Anatomy of the ear, nose and throat, neck, chest, esophagus, and the related nervous systems.
2. Pathology.
3. Bacteriology.
4. Physiology.
5. Didactic otolaryngology.
6. Fundamentals of surgery.
7. History of otolaryngology.
8. Discussions of current literature.

Although no definite time has been determined for the teaching of these subjects the Board has suggested 8½ to 9 months, or an academic year of training.

As an allustration of a formal course the University of Pennsylvania offers in its present course in basic science in otolaryngology:

1. Anatomy, including embryology and cadaver work (199 hours).
2. Physiology (106 hours).
3. Pathology (108 hours).
4. X-ray (9 hours).

5. Didactic instruction on subjects other than the foregoing (396 hours).

6. Sixty-seven hours of work in the dispensary and operating room.

This is approximately an eight months' course.

Columbia University in New York offers to its affiliated hospitals a course in:

1. Dissection of the head and neck.

2. Embryological development of the nasal cavity and the sinuses.

3. Operations, surgery of ear, nose and throat.

4. Histopathology and physiology of the nose and throat.

5. Histopathology and physiology of the ear.

6. Surgical anatomy of the temporal bone.

7. Anatomy of the special senses.

These are not listed as to the number of hours but are given at various times in the afternoon and evening, throughout the year, to accommodate the residents in the affiliated hospitals. These courses are open only to residents in affiliated hospitals.

Selection of Residents. The American Medical Association states that the residents should be selected from graduates of an approved medical school, who have served an internship in an approved hospital or who have two or more years in practice in the specialty of otolaryngology. It is not sufficient that they merely have a surgical internship. In these days of chemotherapy, a great deal of otolaryngological surgery is a thing of the past. This affords the opportunity to increase our knowledge of contributing medical causes in diseases of the ear, nose and throat. This has been a neglected field. It is a large field. Although, at the present time, an applicant for a residency in otolaryngology should have a surgical internship, the time may come when we may well desire a resident with medical, rather than surgical, training.

A resident should have a breadth of vision beyond just surgery in the otolaryngology of tomorrow. The resident should be selected on the basis of his broad cultural and educational background, a good scholastic record at college and medical school, general ability, eager-

ness to acquire knowledge, a real interest in his specialty, integrity, strength of character, and a discriminating sense of ethical values.

Training. The department of otolaryngology should be independent or autonomous from an operational standpoint. It should have its own instruments, equipment, adequate library facilities, textbooks, and journals. The scope of training should include training in oral surgery, plastic surgery, neck surgery, broncho-esophagology, allergy, some types of neuro-surgery, pediatric otolaryngological training and the fundamentals of surgery.

The outpatient department should be sufficient to offer adequate training. It is in the clinic that the resident learns the niceties of the practice of otolaryngology; the simple things of otolaryngology, so frequently poorly done. For instance, history taking, manipulation of instruments, sitting position in examination, the use of the head mirror, the use of the laryngeal and postnasal mirrors, the lavage of sinuses, the proper inflation of the eustachian tubes, what to tell and what not to tell the patient, what to discuss and what not to discuss before them, the local applications of medicine, and refinements of office practice. These refinements are essential in the practice of otolaryngology and make the resident ultimately, as far as the patient is concerned, someone who helps him, who makes him feel better, and in whom he has confidence.

The clinical training in a well-organized otolaryngological service depends directly on how much time the Chief of Service spends with the residents on the wards and in the clinics. It is only through him, who has attained this position because of special merit, that inspiration is imparted to the resident staff and to the subordinate attending staff. There should be a paid instructor in minor surgery and diagnostic procedures, such as hearing testing. Many false premises have crept into a hospital service by both surgical and clinical procedures having been passed on from one resident to another. Clinical conferences, study courses, didactic lectures, and anatomical demonstrations, instruction in bedside manner, handling of private patients and office treatment may well follow the basic science course. One can go on indefinitely enumerating the refinements of resident teaching.

Due consideration should be given to living conditions, remuneration, depending upon the geographic location, and adequate malpractice protection for the resident, while he is attached to the hospital staff.

From these thoughts I think it can be stated that there are few residencies today offering anything like an ideal residency in otolaryngology, from either the resident's viewpoint or the American Board's viewpoint, that the new requirements for 1950 necessitate a complete review and conversion of all residencies during 1947 to meet the new requirements. This is healthy in that it demands such a review. The conversion is going to be very difficult.

At the Manhattan Eye, Ear and Throat Hospital, we are undertaking such a conversion, and we present to you the following conversion set-up. It will be necessary for us to teach our own basic science courses, which we think we can do. We will be prepared:

1. To give a three-year residency complete with basic sciences.

2. To accept residents for two years who have taken their basic sciences elsewhere.

3. To give a certain number of basic science courses to those who want basic sciences in preparation for another residency.

This conversion plan suggests a rotating admission of residents to the staff, so that those who will appear before the Board in 1950, with its increased requirements, will be prepared, and at the same time those already in residency who will appear before the Board in 1947, 1948 and 1949 under the present Board requirements will be kept in line. There is no disruption of sequence in the residencies and no undue hardship placed on the attending or the resident staff.

We have prepared two charts* which illustrate the suggested conversion plan.

In going over material for this paper, amongst various rules for residencies I have found one which was printed in "Hospital Management," September 1946: "It seems as if it would be necessary to have to order interns and residents not to smoke when on duty; not to laugh out loud; not to cut up with the nurses where it may be heard by the patients and visitors; not to pinch nurses where the patients may hear the squeal; and to avoid all those things which do not become a professional gentleman."

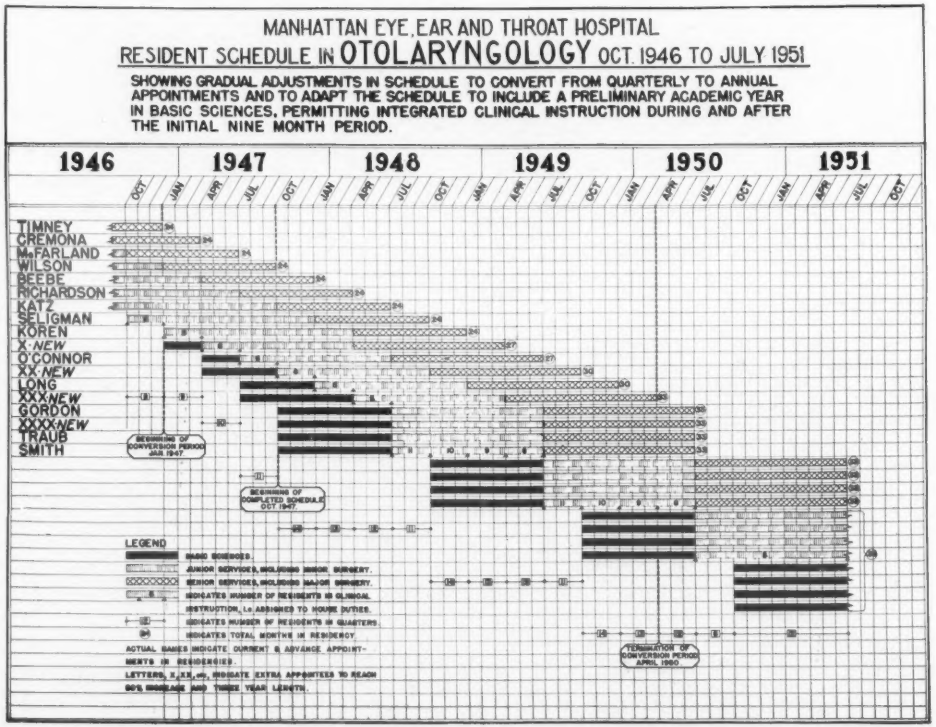
907 FIFTH AVENUE.

*Conversion chart prepared by Mr. Fred Heffinger, Superintendent, Manhattan Eye, Ear and Throat Hospital, New York. Basic science chart prepared by Dr. Marvin Jones, Surgeon-Director, Manhattan Eye, Ear and Throat Hospital, New York.

MANHATTAN EYE, EAR AND THROAT HOSPITAL

OUTLINE OF SCHEDULE IN BASIC SCIENCES IN OTOLARYNGOLOGY

HOUR	OCT.	NOV.	DEC.	JAN.	FEB.	MARCH	APRIL	MAY	JUNE
9-10	HISTOLOGICAL ANATOMY, EAR.	HISTOLOGICAL ANATOMY, EAR.	PATHOLOGY, EAR.	PATHOLOGY, EAR	PATHOLOGY, EAR	PATHOLOGY, EAR	PATHOLOGY, EAR.	PATHOLOGY, EAR.	PATHOLOGY, EAR.
10-11	HISTOLOGICAL ANATOMY, NOSE & THROAT.	HISTOLOGICAL ANATOMY, NOSE & THROAT.	PATHOLOGY, NOSE & THROAT.	PATHOLOGY, NOSE & THROAT.	PATHOLOGY, NOSE & THROAT	PATHOLOGY, NOSE & THROAT	PATHOLOGY, NOSE & THROAT.	PATHOLOGY, NOSE & THROAT.	PATHOLOGY, NOSE & THROAT.
11-12	EMBRYOLOGY, EAR	EMBRYOLOGY, NOSE & THROAT	PHYSIOLOGY & BIOCHEMISTRY, EAR.	PHYSIOLOGY & BIOCHEMISTRY, NOSE & THROAT	PHYSIOLOGY & BIOCHEMISTRY, NOSE & THROAT & EAR	BACTERIOLOGY & IMMUNOLOGY, NOSE & THROAT	BACTERIOLOGY & IMMUNOLOGY, EAR.	BACTERIOLOGY, IMMUNOLOGY & HAEMOTOLOGY EAR, NOSE & THROAT.	a. PHYSICS OF SOUND. b. RADIATION PRINCIPLES. c. ELEMENTS OF ELECTRICITY. d. PHARMACOLOGY.
1-2	PREPARED AND GRADED INSTRUCTION IN-PATIENT DEPARTMENT HISTORIES AND PHYSICAL EXAMS — BEDSIDE TEACHING IN APPLIED BASIC SCIENCE SUBJECTS B. DIRECTORS ON SERVICE — INTERPRETATIONS OF RADIOGRAMS								
2-3	PREPARED AND GRADED INSTRUCTION OUT-PATIENT DEPARTMENT CLINICAL APPLICATION OF BASIC SCIENCES SUPERVISED & TAUGHT BY RANKING CLINICAL TEACHERS — INTERPRETATION OF RADIOGRAMS FUNCTIONAL TESTS, I. ACOUSTIC-AUDIOMETRY-AUDIOLOGY. 2 VESTIBULAR.					DISEASES OF THE NOSE, THROAT AND EAR. PERORAL ENDOSCOPY—ORAL DISEASES AND SURGERY PLASTIC SURGERY-ALLERGY & IMMUNOLOGY.—NEUROLOGY.			
3-4	ANATOMY EAR, NOSE & THROAT. DISSECTION GROSS SPECIMENS. MODELS & MODEL CONSTRUCTION.						OPERATIVE SURGERY ON CADAVER-EAR, NOSE & THROAT. NECK SURGERY-CRANIAL SURGERY-PLASTIC SURGERY-FACIO-MAXILLARY SURGERY-ORAL SURGERY-PERORAL ENDOSCOPY.		
4-5	DAILY RECORDING OF NOTES IN RESIDENTS DAILY LOG AND STUDY PERIOD.								
7-8	JOURNAL CLUB ONCE EACH MONTH — RESIDENT PROGRAM ONCE EACH MONTH. ROUND-TABLE DISCUSSION ONCE EACH MONTH — CRITICS HOUR ONCE EACH MONTH. LECTURES AND DEMONSTRATIONS IN THE FOLLOWING SUBJECTS.								
	1. DISEASES OF THE ORAL CAVITY. 2. RADIUM AND X-RAY THERAPY. 3. ALLERGY. 4. ROENTGENOLOGY. 5. NEUROLOGY.	6. PSYCHIATRY.	7. ANESTHESIOLOGY. 8. SPEACH REHABILITATION. 9. AURAL REHABILITATION. 10. INDUSTRIAL OTOLARYNGOLOGY. 11. EFFECTIVE SPEAKING.		12. MEDICAL WRITING AND ILLUSTRATIONS. 13. HOME CARE OF PATIENTS. 14. OFFICE PROCEDURES. 15. SURGICAL PRINCIPLES. 16. EYE DISEASES ASSOCIATED WITH E.N.& T.			17. ELECTRONICS IN OTOLOGY. 18. RESEARCH METHODS. 19. DISEASES OF THE ESOPHAGUS. 20. DISEASES OF THE CHEST. 21. DERMATOLOGY IN OTOLARYNGOLOGY	


MANHATTAN EYE, EAR AND THROAT HOSPITAL
RESIDENT SCHEDULE IN OTOLARYNGOLOGY OCT. 1946 TO JULY 1951
SHOWING GRADUAL ADJUSTMENTS IN SCHEDULE TO CONVERT FROM QUARTERLY TO ANNUAL APPOINTMENTS AND TO ADAPT THE SCHEDULE TO INCLUDE A PRELIMINARY ACADEMIC YEAR IN BASIC SCIENCES, PERMITTING INTEGRATED CLINICAL INSTRUCTION DURING AND AFTER THE INITIAL NINE MONTH PERIOD.
1946
1947
1948
1949
1950
1951
OCT
JAN
APR
JUL
TIMNEY
CREMONA
McFARLAND
WILSON
BEEBE
RICHARDSON
KATZ
SELIGMAN
KOREN
X-NEW
O'CONNOR
XX-NEW
LONG
XXX-NEW
GORDON
XXXX-NEW
TRAUB
SMITH
BEGINNING OF CONVERSION PERIOD JAN. 1947
BEGINNING OF COMPLETED SCHEDULE OCT. 1947
TERMINATION OF CONVERSION PERIOD APRIL 1950
LEGEND
ACTUAL DATES INDICATE CURRENT & ADVANCE APPOINTMENTS IN RESIDENCIES
LETTERS, X, XX, etc. INDICATE EXTRA APPOINTEES TO REACH 20'S, DOUBLE AND THREE YEAR LENGTH.

VI

THE CLASSIFICATION AND TREATMENT OF PATIENTS WITH CHRONIC NASAL SYMPTOMS

EDWARD D. KING, M.D.

HOLLYWOOD, CALIF.

Sinus disease is not so common as we are led to believe by the large number of patients who complain of nasal symptoms. For a long time headache, especially over the eyes, has been attributed to sinus infection. The sinuses have been accused also in chronic obstruction and dripping in the throat. The rhinologist is responsible to a great extent for this misapprehension because in the past he has failed to classify these cases properly. As a result, therapeutic efforts met with little success in a great many cases and the profession and the public have little faith in measures instituted for the treatment or cure of sinus disease.

Acute sinus infections follow in the wake of a severe cold or influenza and sometimes are the result of a tooth infection. The symptoms and signs are so well known that there is no need to discuss this phase. Here we are concerned with chronic cases which make up the majority of those who complain of nasal symptoms.

A great many patients state that "sinus" is the chief complaint. When these people are questioned closely, the symptoms are disclosed and they consist mainly of nasal obstruction, headache, dripping in the throat and nasal discharge. The history is important. The onset may be ascribed to a cold or influenza; in allergic cases it may be indefinite. It is not uncommon for allergies to follow acute upper respiratory infections. For that reason we go into detail in the history. Patients who have chronic sinus disease as a rule can definitely state the onset. They will give a history of repeated attacks at intervals, generally unilateral. The general history must be considered because of the influence other conditions have on the nasal mucosa. It is not uncommon to see women in the menapause complaining of nasal obstruction and headache. Conditions relating to allergy

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Presented before the Western Section Meeting of the American Laryngological, Rhinological and Otological Society, Los Angeles, Calif., Jan. 26, 1947.

of the nose and sinuses such as asthma, eczema and hives, must be noted and the family history must not be forgotten.

The next thing in order is a smear of the nasal secretions. In patients who complain of postnasal drip or other throat conditions we include a smear of the secretion from the throat. Hansel has devised a stain which is very useful. A slide can be made quickly by his method. In our hands it has proven of great value. The smear will be helpful if interpreted properly. Where infection is present the smear will reveal numerous pus cells with no eosinophiles. Frequently we are unable to differentiate between an acute cold and allergic rhinitis. A smear of the nasal secretion will solve that at once. In allergic rhinitis the smear will reveal a pure eosinophilia. In cases of chronic sinusitis the secretion is much less in quantity but a small amount spread on a slide will aid greatly in reaching a diagnosis because in sinusitis the eosinophiles will not occur whereas in allergic rhinitis the eosinophiles will be found in most cases. We have experienced great satisfaction from the smears made of the throat secretions. We make the smear by picking a small quantity of the secretion from the posterior wall of the pharynx with a long forceps and spreading it gently on the slide. We have explained a good many puzzling throat symptoms by finding eosinophiles in the smear from the throat.

The physical examination is done next. The rhinologist must inspect the nose and nasal pathways, the postnasal space, the mouth, the throat and the ears. If shrinking solutions or cocaine are to be used, it is well to defer them until an x-ray film of the sinuses has been taken. This should be done routinely. This examination should disclose the presence or absence of infection. If clouding of a sinus appears on the x-ray film, it may be necessary to irrigate in order to rule out infection. It is well known that this clouding does not necessarily mean infection. Sometimes a swelling of the mucosa occurs as a result of allergic rhinitis. There are other causes for opacities on the sinus roentgenograms besides infection. It is conceded that x-ray films of the ethmoid sinuses are difficult to interpret. In fact some authorities disregard ethmoid x-rays entirely. If infection is present repeated inspections of this area will reveal it. Much has been said about hyperplastic ethmoiditis, or wet membranes in the ethmoid floor. We are quite sure that these conditions are not the result of infection but of early allergic changes in the mucosa.

Corrections for nasal aeration and proper breathing are essential and the rhinologist must decide whether an operation will remedy

the trouble. It is obvious that large polyps which obstruct the passageways must be removed, dislocation of the end of the septum or collapse of the alar wings may claim his attention and may necessitate an operation. However, the rhinologist must exhaust every means before advising the patient that a simple septal resection will solve the problem. If the patient cannot be helped by means of purely rhinologic procedures and infection of the sinuses has been ruled out, we must turn to the methods which are logical and effective, namely allergy studies.

We perform skin tests to determine the offender. We have used the intradermal tests because over a long period of time we have had satisfactory results. It is not necessary in the majority of patients to do numerous tests. The common inhalants and foods are used for testing and most patients will respond to treatment. Too much emphasis must not be placed on the skin tests. It is a common experience to see allergic patients who have had numerous tests with negative findings. We see patients too frequently who have daily symptoms and yet are told they showed positive skin tests only to some rare or exotic food. This practice throws a bad light on the technique and should be avoided. Their value can be vitiated by attempting to interpret them too closely. Any patient who has symptoms constantly should be protected by a routine survey of the environment, no matter what the skin tests show. This means that feathers and house dust should be eliminated in every case. It is well to inquire into the use of nose drops because all of us have had the experience of eliminating nose drops and finding the symptoms abated.

Many patients who are sensitive to house dust will complain of more severe symptoms in the winter months because house dust is more concentrated when the windows are closed and the furnace turned on. These are the patients who state they have repeated sinus attacks through the winter months. Close inquiry will reveal that the general constitutional symptoms and fever are absent. These cases respond well to small graduated doses of house dust.

Recurrent colds should arouse our suspicions. This is especially true in children. A smear made from the nasal secretion is sufficient for diagnosis. Bacterial allergy is frequently given as a cause of recurrent colds in children. This concept is a result of a failure to make thorough allergic studies of these patients. In our experience bacterial allergy is rare. We feel that studies of the nasal secretions and the environment, with elimination of the offending allergens, will solve most of these cases.

Chronic cough is sometimes the only symptom of allergy. When all other causes have been eliminated, allergy should be considered. We have seen cough eliminated completely by covering a feather pillow or by a few injections of house dust. During the pollen season cough may be the only symptom. It has helped us a great deal to have the patient cough up some secretion and to make a smear for eosinophiles. Mistakes in diagnosis will be avoided if a careful history is taken and slight signs of allergy, such as a mild eczema or small polyps in the nasal mucosa, are carefully noted.

Dripping in the throat is a common complaint and in our experience allergy is the most common cause. It is admitted that the symptom is given too much emphasis by the patient and sometimes by the doctor. Yet it is annoying. By desensitization or elimination diets we have been able to prove to the patient that this symptom can be controlled.

Allergy is becoming more common. Many industrial allergies are appearing as a result of development of new products in industry. Many patients with pollen allergy will develop other allergies after a few years. It is no problem to recognize the case that manifests the typical signs of allergy during the pollen season. The border line cases are the ones that offer problems in diagnosis and they present a constant challenge to the rhinologist. The patient with chronic symptoms may need a submucous resection or the removal of large obstructing polyps but the fundamental cause of the symptoms may not be touched by these procedures. The history, the examination and the treatment must be well correlated before results can be obtained.

6777 HOLLYWOOD BLVD.

VII

THE BASIC PRINCIPLES OF RADIATION THERAPY

LEWIS FRANCIS MORRISON, M.D.

SAN FRANCISCO, CALIF.

The welfare of the patient who has a cancer of the upper respiratory tract is the background for this paper. Advances in radiation therapy have produced results that permit a certain latitude in the selection of therapeutic agents. There was a time when one was obliged to employ surgical measures when there was reason to believe that the entire growth could be removed. This is no longer obligatory. Facts and figures are available to substantiate this statement. The criteria for the selection of a therapeutic agent—surgery or radiation—do not come under the scope of this paper. The basic principles of radiation therapy are offered with the sincere hope that the facts as presented will accomplish two things: first, to encourage the otorhinolaryngologist and the radiologist to speak a common language; second, to stimulate an interest in the advance in nuclear physics.

Otorhinolaryngologists in general have little conception of what has transpired in the realm of physics since they were subjected to the dry, cold facts in the premedical years. An extensive knowledge of higher mathematics is not essential. The newer phases are fascinating, offer intriguing possibilities and fulfill the demands of the imagination as well as the proven practical side. These points are guaranteed.

The future of the patient with cancer of the upper respiratory tract depends on the knowledge of his medical adviser. This makes it imperative that the adviser have access to and employ such knowledge as is available.

An accurate diagnosis is essential. The histopathological description must be conclusive. A diagnosis of cancer made entirely on clinical findings is unfair to the patient and deliberately un-

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Read before the Western Section Meeting of the American Laryngological, Rhinological and Otological Society, Los Angeles, Calif., January 25, 1947.

scientific. At times repeated biopsies are necessary. An equivocal diagnosis is not acceptable. In most instances one can be quite certain as to the nature of the visible lesions. This proximity to accuracy is not enough. The decisions are all-important to the future of the patient. They must depend on specific information and that information must be obtained from biopsy material. A patient referred by another physician frequently arrives with a letter stating that he has a malignant growth. Less frequently a copy of the pathologist's report accompanies the letter. Examination may show a lesion that simulates the asserted diagnosis. In all instances the otorhinolaryngologist and the radiologist should either obtain the original slides for verification or insist that the diagnosis be re-established by another biopsy. This attention to detail will cause some annoyance at times but an accurate diagnosis is necessary before one can discuss prognosis or intelligently select the type of therapy best suited to the individual.

The segregation of the therapeutic agents into two main categories seems advisable. Surgery is included in both in spite of the fact that the subject matter under discussion is primarily radiation therapy. It is included to remind one that there still exists a quick, clean, direct method that, in properly selected cases, does effectively and efficiently remove the malignant growth from the host. Also there are instances wherein both surgery and radiation therapy produce better results than either used alone.

The segregation of the agents is arbitrarily:

A. Those of the past
   1. Surgery
   2. Radium
   3. Radon—unshielded glass capillary tubes and later gold capillary tubes.
   4. Low voltage x-ray (massive dosage).

B. Those of the present
   1. Surgery
   2. Radium
   3. Radon in shielded capillary tubes
   4. X-ray (200 K.V. and super voltage—fractional dosage).
   5. Cyclotron—Neutrons, radioactive isotopes, mesons and the yet unexplored therapeutic value of highly activated alpha particles.

Fig. 1.

The relative positions and sources of the radioactive agents is presented in the accompanying illustration (Fig. 1). The diagram is simple. In some respects it needs no explanation; in other respects volumes would be necessary. It is adequate as a graphic representation of today's knowledge.

Mode of Action of the Agents. Surgery (sharp dissection, cautery, electrocoagulation).

The underlying principle of mechanical removal of the neoplasm plus sufficient normal tissue to preclude recurrence is well known and does not warrant elaboration at this time.

Radioactive Agents. The therapeutic action of all radioactive agents depends on one physical property—the ability to produce ionization. Ionization is the direct result of expenditure of energy from the radiation source in the exposed tissues. This results in a disruption of the intracellular molecular arrangements or bonds that are essential to the life cycle of the cell. The control of quantity or degree of ionization in relation to time and space is all important.

Present knowledge leads us to believe that the nucleus of the cell is more vulnerable than the cytoplasm. It also allows the conclusion that the nucleus is most vulnerable during the stages of mitosis. This accounts for the marked response to therapy in rapidly growing neoplasms. This fact allows a sufficient margin of safety between de-

struction of the normal cells of the host and those of the neoplasm to permit lethal radiation of the malignant cells without complete destruction of the normal tissue cells. The histopathological picture substantiates this view. Pyknosis and karyolisis are more evident in the neoplastic cells. The margin of safety is well illustrated by the good results obtained by prolonged fractional radiation therapy as contrasted with those following mass doses wherein the margin of safety was contracted to a very thin line.

A converse way of explaining the results is frequently employed. The gist of the explanation rests on the statement that the recovery factor of the normal cell is greater than that of the neoplastic cell. The conclusion that there is a difference in vulnerability between the malignant and normal cells is the same.

There is not at the present time any agent whose selective action is such that only aberrant cells are destroyed and the adjacent normal cells are left wholly unscathed. In other words, the elimination of a neoplasm necessitates the sacrifice of a certain number of the normal cells of the host. This explains the reactions during and subsequent to radiation therapy. Mucositis and epithelial reactions must be anticipated and expected. These reactions are component parts of radiation therapy of the present day and should not be considered as "burns". Postradiation edema and varying degrees of pigmentation change, telangiectasia and permanent epilation of the treated areas must be expected. The vain hope of some radiologists to avoid or minimize these reactions and the possible medicolegal complications by deliberate inadequate tissue dosage has been costly to both the patient and the progress of radiation therapy.

There is one gross difference between the surgical removal of a neoplasm and the successful elimination of a neoplasm by radiation therapy. When surgery is employed the neoplasm is mechanically removed. This necessitates the removal of varying amounts of normal tissue and in some instances the removal of important functional structures. Radiation therapy accomplishes the removal of the neoplasm with a minimal loss of normal tissue. This is accomplished in situ and usually entails no loss of function of the structure. This destruction of tissue in situ necessitates the scavenging and elimination of a considerable amount of foreign protein. This undoubtedly accounts for much of the general systemic reaction that accompanies radiation therapy. The local reactions are readily understandable. The general reactions are more difficult to explain. The knowledge that these local and general reactions are

inevitable emphasizes the importance of conditioning the patient both mentally and physically prior to, during and after the institution of radiation therapy. These phases are as important to the welfare of the patient receiving radiation therapy as are the similar phases when surgery is employed.

If one is to expect any degree of cooperation the patient-doctor relationship must be complete. There is only one way to obtain this relationship and maintain it throughout the trying periods during the radiation reaction peaks and the years that follow. Total and absolute honesty is the only bond that is strong enough to keep this relationship intact. This frankness applies to diagnosis, prognosis and expected discomforts both during and following therapy. These facts must be discussed with the patient as soon as the diagnosis is definite and before radiation therapy is started. In some quarters there is some controversy as to the advisability of telling the patient that he or she has a cancer. As far as I am concerned there are no grounds for controversy. In spite of the emotional reactions of the family and relatives, the patient is the all-important individual and should know what is wrong and what can be done about it. I have followed this practice without deviation and am not able to report a single instance wherein the commonly assumed psychic trauma induced the patient to resort to self-destruction. Contrarily, it has resulted in a closer patient-doctor bond. The patient then feels assured that there is at least one person from whom he can expect a direct and honest answer. Many questions cannot be answered directly because present available knowledge has not supplied the material for such an answer. In these instances one can only answer "I do not know". Such an answer does not undermine the patient's confidence.

The ability of the otorhinolaryngologist and the radiologist to speak a common language and to have sufficient knowledge to permit a moderate understanding of one another's problems are essential. This is not asking too much of either party. It is vital for the patient's welfare. The otorhinolaryngologist should have more than a nebulous idea as to what is meant by an "r." Even the knowledge that it is an arbitrary unit, defined as the amount of radiation energy necessary to ionize one cubic centimeter of air under standard conditions of temperature and pressure is not sufficient. He should be mentally equipped to ask at least semi-intelligent questions in regard to the details of the technique employed by the radiologist. The mere statement that the patient received so many thousand "r"'s means nothing. The literature abounds with articles containing these cryp-

tic statements. The intimate details as to the source's ouput in r's per minute, filtration factors, target distance, size of port or field treated do become a bit cumbersome, mathematically speaking, to those who are not primarily interested in the physics of radiation. The whole problem could be simplified by confining the statement to the all important factor—tissue dosage. Every competent radiologist bases therapy on this factor. Few otolaryngologists are equipped to carry out the necessary mathematics and there is no real reason why they should so equip themselves. They should know that there is a difference between roentgens measured in air and those in tissue and that some of this difference is due to back scatter as well as just a difference in medium. The dose the selected tissue or tissues receives is the all important factor. This holds true whether one is dealing with roentgens, neutrons or millicurie hours, and will obtain with other forms of radiation therapy when such are available.

The knowledge obtained in learning to speak a few sentences in the radiologist's language should not be permitted to unbalance the otorhinolaryngologist to the precarious position where he decides that he is competent to outline and direct the course of radiation therapy. No competent radiologist would be a party to such stupidity. Any such request is both obnoxious and insulting. The radiologist is not just a trained technician. Discussions between the radiologist and the otolaryngologist as to the factors of time, total tissue dosage, size of field, and reaction should be welcomed. They are stimulating to both parties and of definite advantage to the patient. Owing to individual variations, no two cases are exactly alike. This precludes the fixed adherence to any so-called "method." Even superficial perusal of the articles written by the men whose names have become associated with methods brings out the facts that a certain routine can be expected to produce certain results. The suggested routines are offered as base lines to obviate the repetition of fruitless trials and errors. All recognize some individual variation.

Thus far the suggestions in regard to learning a common language have been confined to the otorhinolaryngologist. They will be left that way except for one remark. It is truly a pleasure to work with a radiologist who is not only able to watch the reactions in the larynx, pharynx and nasopharynx and other visible areas in our field but actually does so and finds time to discuss the details with the referring otorhinolaryngologist.

CONCLUSIONS

1. The basic principles of radiation therapy have been presented under the five headings: diagnosis, therapeutic agents, mode of action of these agents, patient-doctor relationship, and the necessity of a common meeting ground for the otorhinolaryngologist and the radiologist.

2. At the present time there is no radiation agent available that will eliminate malignant tissue cells without destroying some of the normal cells of the host.

3. The basic principle of the action of all the agents depends on the expenditure of the energy of the agent in intracellular ionization.

4. A close patient-doctor relationship is essential to obtain and maintain the patient's cooperation. An honest discussion of the facts is essential to initiate and perpetuate this relationship.

5. The patient subjected to radiation therapy should have care prior to, during and following treatment equal to that considered necessary for patients who are to undergo surgery.

6. The ability of the otorhinolaryngologist and the radiologist to speak a common language is essential for their own benefit as well as that of the patient.

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VIII

THE USE OF STREPTOMYCIN IN THE TREATMENT OF DIFFUSE EXTERNAL OTITIS

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Evidence has been presented in previous reports of the frequent occurrence of certain gram-negative organisms in the ears of patients with specific types of external otitis. In a very high percentage of infected ear canals, diagnosed as diffuse external otitis, it has been possible to isolate cultures of Pseudomonas* organisms.[1-3] Such organisms could not be obtained consistently from either the contralateral unaffected ear or from the ears of subjects free of ear disease. Similar findings have been reported by other observers.[4-8]

The high degree of correlation between the presence of Pseudomonas organisms in an ear canal and the existence of certain types of external otitis suggests some relationship between these organisms and the disease. Although it has not yet been possible to obtain experimental evidence of the nature of this association, at least two possibilities merit serious consideration. These possibilities are, firstly, that the Pseudomonas organisms are, in fact, the true etiological agents of the disease or, secondly, that they act only as unique secondary invaders and merely aggravate a pre-existing disease condition caused by an as yet undemonstrated agent.

Such considerations are of clinical importance. Owing to its as yet undetermined cause, treatment of this type of external otitis has been nonspecific in nature and the lack of uniform success of such nonspecific treatment is well recognized. On the other hand, if Pseudomonas organisms act either as the causative agents or even

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*Throughout this paper, no attempt has been made to characterize the various species isolated. For the purposes of this study, Pseudomonas will be understood to include all gram-negative motile rods which grew well on ordinary nutrient media and which produced a characteristic water-soluble, chloroform-soluble, greenish pigment.

as secondary, aggravating invaders, it would appear feasible and desirable to substitute specific chemotherapy for nonspecific, symptomatic treatment. Thus, treatment of diffuse external otitis would be aimed at eradication of either the cause or the aggravator of this disease.

Streptomycin is known to be an effective antibiotic substance, in vitro, against many strains of Pseudomonas[9, 10] and has been used with some success in the treatment of infections caused or aggravated by this organism.[11, 12] Accordingly, it appeared of interest to investigate the effect of streptomycin therapy on the above-mentioned type of external otitis.

For this purpose, a series of patients with diffuse external otitis was treated by both topical and systemic streptomycin therapy and an attempt was made to correlate the in vitro sensitivity of the isolated strains of Pseudomonas to streptomycin, with the clinical response of the disease to therapy.

Methods. Fifty-four consecutive patients with findings which conformed to our criteria for diffuse external otitis[1, 2, 3] were obtained from the neighborhood of San Antonio, Texas, during the summer of 1945. After routine medical and otologic examinations, cultures were made from both affected and non-affected ears. In order to compare the bacteriological flora obtained from the ears of these patients with normal ears during the same season, cultures were made from the ear canals of 20 subjects without ear, nose or throat complaints (40 ears) and the organisms identified.

Patients were treated daily and followed with appropriate clinical and bacteriological post-treatment examinations. Whenever it became evident that therapy was producing unsatisfactory results, notations of this fact were made and further treatment with streptomycin was not attempted. Except where noted, all patients were treated on an ambulatory status.

Whenever gram-negative organisms were isolated and identified, a test was made to determine their in vitro sensitivity to streptomycin. This test consisted of streaking a loopful of a six-hour culture of the organism in 2% tryptone onto the surface of a series of plates of 2% tryptone agar, each plate of which contained graded quantities of streptomycin. The plates were incubated overnight and observed the next morning for the presence or absence of visible growth. The sensitivity of the organism was defined as the smallest

quantity of streptomycin required to inhibit growth under these conditions.[13]

In order to evaluate the effects of topical therapy, groups of patients were treated with varying concentrations of streptomycin in an appropriate ointment base and the effects of the treatment with the varying concentrations of streptomycin were compared with those obtained in similar patients from use of the ointment alone (without added streptomycin) and with no treatment other than simple cleansing and drying.

These groups of patients were divided into the following categories of treatment:

1. Streptomycin in "Carboprop"* (250 micrograms of streptomycin per gram) was applied freely to the entire surface of the external auditory canal and a cotton wick impregnated with this ointment was inserted into the canal and allowed to remain overnight.

2. Streptomycin in "Carboprop" (1 milligram of streptomycin per gram) was used in a similar manner.

3. Streptomycin in "Carboprop" (5 milligrams of streptomycin per gram) was used in a similar manner.

4. "Carboprop" alone was used in a similar manner. This group of patients constituted the control for the effects of the ointment alone.

5. The ear canal was cleansed carefully with the aid of hydrogen peroxide and a dry cotton wick was then inserted. This group of patients constituted the control for the effects of simple cleansing and drying.

6. For the study of the effects of systemic therapy, 100 milligrams of streptomycin were administered intramuscularly every three hours for three days for a total of 2.4 grams.

In patients with bilateral external otitis, whenever clinical conditions permitted, one of the affected ears was used as a control (either category four or five above) while the other affected ear was treated with streptomycin ointment. By means of the establishment of such controls, it was believed feasible to evaluate the effect of the antibiotic on diffuse external otitis.

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*60 parts of carbowax 4000 and 40 parts of propylene glycol.

RESULTS

A description of the results follows:

I. *The Bacterial Flora of Normal Ears* (20 normal subjects—40 normal ears): The various types of organisms isolated from these ears indicated that the normal flora in this climate and season consisted principally of various strains of Staphylococcus (both pigmented and unpigmented), various members of the Micrococcus group, diphtheroids and occasional non-hemolytic streptococci; gram-negative rods were isolated from one ear only.

II. *The Bacterial Flora of the Ears of Patients with Infected Ears, Prior to Treatment* (53 patients—106 ears):

A. Affected Ears: All infected ears in this series showed gram-negative rods as the predominant organisms; by far the largest percentage of ears showed cultures of Pseudomonas organisms. Occasional strains of Alkaligenes, Aerobacter and Proteus were identified.

B. Nonaffected Ears: With few exceptions, gram-negative rods were not obtained from the nonaffected ears. Even when a relatively pure culture of Pseudomonas was obtained from the affected ear, the uninvolved ear would demonstrate only staphylococci, micrococci, streptococci and diphtheroids.

III. *Bacterial Flora of the Ears of Patients with Infected Ears, After Treatment*:

A. Affected Ears:

1. Streptomycin, 250 micrograms per gram: Six of nine cases showed a persistence of Pseudomonas after one to five days of therapy.

2. Streptomycin, 1 mg. per gram: Five of eight cases showed a persistence of Pseudomonas after two to five days of therapy. The culture plates contained fewer colonies per plate than before treatment.

3. Streptomycin, 5 mg. per gram: Nine of ten cases showed complete disappearance of Pseudomonas after one to five days of treatment.

4. Systemic streptomycin therapy: Pseudomonas persisted in pure culture in both ears of the treated case.

B. Nonaffected Ears: The bacterial flora of nontreated, nonaffected ears remained essentially unchanged during the period of observation. In two ears, gram-negative organisms which had previously been present disappeared during observation. Several nonaffected ears which showed Pseudomonas on culture but no symptoms or signs of external otitis developed acute diffuse external otitis while still under observation.

IV. *Clinical Response of Infected Ears Following Treatment*:

A. With Streptomycin Locally.

1. 250 micrograms per gram (nine patients): Two cases showed increasing pain and edema; three cases improved temporarily, but subsequently relapsed; two cases showed little improvement; two other cases evidenced an excellent response with rapid improvement.

2. 1 mg. per gram (nine patients): Poor results were obtained in four cases; five cases showed a good clinical response without recurrence.

3. 5 mg. per gram (13 patients): Seven cases showed a rapid response and six others improved slowly but definitely. No failures or recurrences were noted in this group of patients. In general, it appeared as if the better results were obtained with the less severe cases.

B. With Streptomycin Parenterally (one patient):* Little improvement occurred during three days of therapy.

C. With "Carboprop" Alone (nine patients): One patient with severe, diffuse external otitis required hospitalization for control of pain; three cases showed no improvement. Five other cases showed improvement with rapid subsidence of symptoms.

D. With Dry Cotton Wicks (14 patients): Eleven cases showed increasing severity of symptoms; in seven of this group, the increasing pain required opiates for control. In three cases, symptoms subsided but recurred after four to six days.

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*Because of the limited supply of streptomycin available at the time of this study it was not possible to increase the number of patients in this category.

V. *In Vitro Sensitivity of Pseudomonas Strains Isolated From Ears*: The majority of isolated strains, as determined by duplicate tests, was sensitive to 10 micrograms of streptomycin per ml. A few required as much as 20 micrograms and an occasional strain was inhibited by as little as one microgram. No detailed analysis is appended but it was impossible to obtain any correlation between clinical response to streptomycin and the in vitro sensitivity of the isolated strain.

DISCUSSION

It is of interest to consider the above results as they apply not only to the treatment of external otitis but also to the relationship between Pseudomonas and this disease. In the first place, previous observations of the close association between this type of disease and this organism have been amply confirmed by the repeated isolation of Pseudomonas from the ear canals of patients with external otitis and its relative absence from the ears of normal patients.

In the second place, the results of this study indicate a high degree of correlation between the ability of an agent to promote the disappearance of Pseudomonas from an infected ear canal and its ability to effect a clinical cure. In general, a review of the results indicates that the use of concentrations of streptomycin ineffective in producing a high percentage of cures was not followed by the rapid disappearance of Pseudomonas in cultures taken from the treated ear. On the other hand, the use of the concentration of streptomycin (5 mg. per gram) found to be most effective clinically resulted in the rapid disappearance of Pseudomonas from such ears.

In a few instances, although treatment apparently resulted in clinical cure, Pseudomonas continued to be present in cultures from the treated ear. In such cases, cessation of treatment was frequently followed by relapse. Furthermore, in one instance in which Pseudomonas was cultured from the ear of an apparently normal subject, such an observation was followed shortly by the development of an external otitis in the ear canal from which the culture had been obtained.

As a result of earlier studies[1, 2] it was concluded that Pseudomonas was not present in normal ear canals but was present in a very high percentage of infected ear canals. The present observations emphasize even more strongly the close association between Pseudomonas and this disease since they demonstrate that the transition of a normal ear canal to a diseased one is accompanied by the appear-

ance of Pseudomonas in canal cultures, and, conversely, that the transition of a diseased ear to a normal one is accompanied by the disappearance of this organism from the canal.

Among the possible associations considered between Pseudomonas and external otitis was that this organism, while not the actual cause of this disease, acted in the role of an aggravating secondary invader. Our present observations in regard to the presence of Pseudomonas in the ear canal, both prior to the development of clinical external otitis and prior to relapse following treatment, strongly hint that the presence of Pseudomonas in an ear canal precedes rather than complicates the development of the clinical syndrome under discussion. Such a conclusion need not necessarily invalidate a hypothesis that preliminary subclinical disease is essential for the development of diffuse external otitis associated with Pseudomonas. Indeed, if such a hypothesis is true, attempts to reproduce clinical external otitis by mere introduction of Pseudomonas into normal ear canals must be doomed to failure and it may help explain the comparative rarity of this disease in spite of the excellent opportunities for the dissemination of Pseudomonas.

In so far as the clinical results of therapy are concerned, the two lower concentrations of streptomycin (250 micrograms per gram and 1 mg. per gram) produced effects no better than those obtained from the use of the Carboprop ointment base alone. It seems apparent, therefore, that such concentrations of streptomycin are ineffective. This is not surprising since the use of streptomycin in conventional dosages for the treatment of other types of local infections associated with Pseudomonas has not always met with the anticipated degree of success.[12, 14] In this particular series, failure of treatment was not associated with resistance of the organism since all of the isolated strains were sensitive, by in vitro tests, to concentrations of streptomycin far less than those applied.

On the other hand, treatment with 5 mg. of streptomycin per gram gave results definitely superior to those obtained with the ointment base alone. The results of this type of therapy, while not ideal, are certainly promising and warrant further investigation as soon as this antibiotic becomes more readily available. They indicate that such concentrations of streptomycin produce a definite clinical benefit and that their use will be attended with success in cases associated with sensitive strains of this organism.

The comparative success of the highest concentration of streptomycin contrasted with the failure of the two lower concentrations

is somewhat reminiscent of local penicillin therapy where concentrations of penicillin are required which are far in excess of those calculated from in vitro sensitivity data. In the case of external otitis, peculiar anatomic considerations and the presence of cerumen may possibly account for the need for such high concentrations of streptomycin. The failure of intramuscular therapy, although limited to one case, was not altogether unexpected since there was no assurance that the relatively low blood concentrations of streptomycin resulting from this mode of administration would be available in effective quantities to the diseased tissues.

Analysis of the results indicates that some benefit was obtained in those cases treated with Carboprop alone, although in vitro studies by the cup plate method revealed that neither propylene glycol nor carbowax 4000 nor mixtures of the two displayed marked bacteriostatic activity against ten of the isolated strains of Pseudomonas. However, it is well known that almost any nonirritating soothing agent produces some temporary benefit in the treatment of mild cases if the canal is first cleansed and dried thoroughly.

Simple cleansing and drying of the canal failed to produce lasting beneficial clinical results and we were thus not able to agree with the much quoted report that simple dry cleaning of the canal in external otitis suffices to cure the majority of these cases. In diffuse external otitis, active treatment is necessary both to control the symptoms and to prevent exacerbation of disease.

SUMMARY AND CONCLUSIONS

1. Clinical and bacteriological studies were made on the ears of normal subjects and of patients with diffuse external otitis.

2. Pseudomonas was not isolated from normal ear canals. However, this organism was found in a very high percentage of cases of diffuse external otitis.

3. Successful treatment of external otitis was accompanied by a rapid disappearance of Pseudomonas from infected ears. Persistence of Pseudomonas occurred in cases of treatment failure or preceded imminent relapse subsequent to cessation of therapy.

4. The close relationship between Pseudomonas and diffuse external otitis is discussed.

5. Treatment of external otitis with 250 micrograms and 1 mg. of streptomycin respectively per gram of ointment base was no more effective than treatment with the ointment base alone.

6. Treatment with 5 mg. of streptomycin per gram of ointment base gave promising and beneficial results.

7. Simple cleansing and drying of the ear canal failed to produce beneficial clinical results.

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IX

THE HEMORRHAGIC DIATHESES ENCOUNTERED MOST FREQUENTLY IN OTOLARYNGOLOGY

DIAGNOSIS AND MANAGEMENT

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It is not uncommon for practitioners of otolaryngology to encounter during the course of their practice several patients who have a hemorrhagic diathesis. Some of these cases are extremely difficult to classify. Much confusion may be encountered in determining whether a patient is suffering from hemophilia or one of the various forms of purpura such as idiopathic, familial or symptomatic. Treatment is difficult if a correct classification cannot be made.

The two cases cited in this paper occurred in the same family. These patients were thought at first to be true hemophiliacs, but as the results of the blood work became known, it was obvious that we were dealing with a purpura, probably of the familial or symptomatic type.

A discussion of the differential diagnosis of the various forms of purpura will be undertaken in the forepart of the paper. The cases will then be cited and their differential findings discussed.

Certain laboratory tests must be made in all cases where there is a suspicion of a hemorrhagic tendency.

The blood coagulation time in hemophilia is always prolonged. It has been pointed out by some authors that the reports of spontaneous remission of the disease are due to technical failures in the estimation of the blood coagulation time. The following is a technique described by Kark.[1]

Five cc. of blood are drawn from the arm veins in a syringe that has been previously rinsed with physiological salt solution. Care must be taken in making the venal puncture. No air or bubbles must enter the syringe. After removal of the needle from the vein,

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the needle is removed from the syringe and 2 cc. of blood are introduced gently, and without bubbles, into each of two 100 x 13 mm. test tubes that are very clean. The tubes are then kept at a temperature of 37° C. The first tube is observed from time to time by turning it gently. The end point is the time at which the surface of the blood has jellied sufficiently to permit inverting the tube without loss of its contents. The second tube is then examined similarly. If the time discrepancy between the two tubes is no greater than five minutes, the average time is used. If the difference is greater than five minutes, the longer time is used.

The tourniquet test is carried out by the application of a tourniquet to the upper arm, which results in the formation of petechiae on the forearm in cases of purpura.

Other laboratory diagnostic procedures that should be done are a platelet count, tests of the bleeding time, the blood prothrombin level, and the clot retraction time.

Farrar and Roxby[2] make the statement that in general one or more of the following defects exist in patients with purpura:

1. Defects of the capillary bed, as in scurvy and nonthrombopenic purpura.

2. Platelet defects, as in symptomatic or idiopathic thrombopenic purpura.

3. Coagulation defects, as in obstructive jaundice or hemophilia.

We hear very much about idiopathic thrombopenic purpura and relatively little about the other types of purpura, when, in truth, the idiopathic type is seen much less often than the symptomatic or secondary types of thrombopenic purpura. The reason for this overemphasis is probably due to the fact that these patients respond so splendidly to splenectomy.

Clinical Findings. The hemophiliac presents the problem of intractable bleeding from an open surface which may have resulted from some accident or some operative procedure. There are some hemorrhages in the subcutaneous tissues and hemorrhages may occur in the joints on the slightest trauma. Mention has been made before of a greatly prolonged coagulation time. The bleeding time is not prolonged and the platelets are normal. The fibrinogen, prothrombin, and calcium are normal in the hemophiliac and the platelets are normal in number. This disease starts in infancy and continues throughout life.

Clinically the forms of purpura may be quite confusing so a brief summary of their clinical findings under the various names will be given.

Meakins[3] states that idiopathic thrombocytopenic purpura is a disease characterized by repeated crops of purpura. These may vary in size and may cover large areas. They may be found on the skin, mucous membrane of the nose, mouth, gastro-intestinal tract, uterus, vagina, urinal tract, and other viscera. The most characteristic sign, by far, is the presence of hemorrhagic spots or areas.

The most specific laboratory findings are:

1. Marked reduction of blood platelets. (Meakins says that when the platelet count gets below 60,000 per cubic millimeter, hemorrhages can be expected).

2. Delayed bleeding time.

3. Decreased contractility of the blood clot (but the clotting time is normal).

4. Positive tourniquet test.

Meakins also mentions a type of purpura which he calls familial, that is, it runs in families. This type does not correspond either to thrombocytopenic purpura or to hemophilia. The hemorrhages may occur in either sex. There is a prolonged bleeding time and clot retraction may be prolonged, but the clotting time and the platelet count are normal. Meakins further makes the statement that many cases do not run true to form, that thrombocytopenia may occur. The hemorrhages are not usually severe and in many cases the tendency to hemorrhage disappears at puberty.

Symptomatic thrombopenic purpura is characterized clinically by sudden spontaneous bleeding, such as epistaxis or postoperative hemorrhage. The clot retracts very slowly and sometimes the tourniquet test is positive. This condition may be caused by drugs, infection, blood dyscrasia, allergy, or endocrine factors.

We see, therefore, that there is some confusion in the diagnosis and definite grouping of cases of so-called purpura.

REPORT OF CASES

CASE 1.—The first patient, C. P., 55 years of age, consulted us on November 17, 1942, complaining of a nosebleed that had been almost continuous for the past ten days, never severe but continually

oozing so that he had to change the cotton packs (inserted by himself) several times during the day.

A small area of bleeding was noticed in the region of Kiesselbach's area. It was cauterized by actual cautery and a small pack of tincture of benzoin-treated gauze was inserted. Two days later he reported that the bleeding had decreased, but that there was still a slight persistent ooze. One day later he reported that all bleeding had ceased. He was not seen again until December 25, 1942, when he reported that the bleeding had not entirely stopped, that every day there were blood stains on his handkerchief. He had not worried particularly about the condition but when he began to feel weak, he asked for further consultation. Hospitalization was suggested but he refused. We saw no more of him until August 22, 1943, that is, about a year later. This time he complained of a nosebleed of several days' duration. The nose was lightly packed with tincture of benzoin-treated gauze and all bleeding seemed to cease. On August 24, 1943, he said the bleeding had increased. He was advised to go to the hospital but refused again as he had done one year previously. He finally entered the hospital upon our insistence on that same day.

At the time of entrance his red blood cell count was 2,680,000; his hemoglobin 54%; and his clotting time 4 minutes, 30 seconds. His blood type was Group 4. He was immediately placed on liver extract, 1 cc. a day; calcium gluconate, 60 grains a day; and hemostatic globulin used as a nasal spray. Morphine, grains 1/6, was given as needed for sedation. When all these measures failed to stop the very slight but persistent oozing from the area, Synkamin* (one ampule every three hours for three doses) was tried with no appreciable effect upon the oozing from the nasal mucosa. The patient seemed to feel well; he did not complain of weakness or tinnitus; his blood pressure remained at 118/85. Three days after admission his red blood cell count was 2,680,000; his white cell count was 7,200; and his hemoglobin 54%. His clotting time remained at 4 minutes, 30 seconds. On September 3, his red blood cells numbered 1,760,000; his hemoglobin was 36%. The tourniquet test was negative.

The results of some of the more special laboratory tests were these: the platelet count was 60,000; the prothrombin time was 27 minutes; the contractility of the clot was very markedly prolonged.

Arrangements were made for a blood transfusion. The following day he received 250 cc. of whole blood. The next day he received

*A synthetic preparation of vitamin K.

another 250 cc. of whole blood. The bleeding by this time had ceased. Two days later he was discharged from the hospital with a red blood cell count of 3,230,000 and a hemoglobin of 66%.

One week after discharge from the hospital his red cells numbered 3,270,000 and his hemoglobin remained at 66%. He was placed on 1 cc. of liver extract every other day and received five doses. He again dropped out of sight.

We were very much handicapped by the fact that the patient was separated from his family when he was nine years old, because of the death of his mother from what he said was tuberculosis. He gave a history of having had transfusions two times previous to the admission to the hospital—once following a tooth extraction several years previously and once again when he had epistaxis two years ago. He said almost all scratches or very slight cuts would bleed slowly for as long as two or three weeks. At one other time after the extraction of a tooth he continued to bleed for almost six weeks. He had become rather accustomed to these bleeding spells. This explains his apparent disregard of any medical attention or follow-up as long as he seemed to feel well and did not become too weak. Moreover, he had been told that he was a hemophiliac. He had become very much concerned that his present employer (should this knowledge be obtained by him) would dismiss him. Hence he had actually avoided medical advice. This added to our difficulty in obtaining further blood work or getting follow-up procedures.

We were able to contact him about two years later on September 10, 1945. He had had a few spells of bleeding for as long as two or three weeks from small scratches obtained while he was working at his position as a janitor. He had become so accustomed to this type of injury that he thought nothing of it. At that time his red blood cell count was 4,100,000; his white blood cells numbered 4,200; his hemoglobin was 75%. His platelet count remained at 60,000; his clotting time was 3 minutes, 30 seconds; but his prothrombin time was still 27 minutes.

CASE 2.—The second patient, R. B., is the grandson of the patient just cited. R. B.'s mother is the daughter of the first patient, C. P. This 20-month-old patient was brought to us for the first time on May 28, 1945. The mother gave a history of the baby having fallen two weeks previously and cutting his upper lip. Since then there has been a slow continual ooze from this region. The mother was advised to take the child to the hospital immediately, which she did.

The child was admitted at 6:30 a.m., May 29, 1945. Examination disclosed the area which was bleeding to be a portion of frenulum of the upper lip. The bleeding was in the form of an ooze and was very persistent. Bleeding continued in spite of the use of silver nitrate or Monsel's solution. The child was given 1 cc. of Koagamin* at 7:00, 9:00, 10:00, and 11:00 a. m. Six drops of paregoric were given at 8:00 and 10:00 a. m. Four hours after admittance all bleeding from the area had ceased. The mother insisted upon taking the child home the following day and against all advice proceeded to do so.

Four days later at two o'clock in the morning she called by phone saying that the baby had started to bleed again. Hospitalization was refused by her. She was then advised to apply a pressure bandage to the area. The next day she called to report that bleeding had ceased. Since then we have not been able to contact the parents. The child's grandfather informed us that there has been no bleeding since we last saw the child.

Laboratory work at the hospital disclosed a red blood cell count of 2,830,000; a white blood cell count of 11,500; hemoglobin 54.4%; a clotting time of 4 minutes; bleeding time of 1 minute. The prothrombin time was 13 seconds; the platelet count 75,600. The tourniquet test was negative.

Treatment of Hemorrhagic Diatheses. It would seem proper at this time to discuss the treatment of the diatheses just mentioned.

In the treatment of hemophilia according to Farrar and Roxby[2] the therapeutic employment throughout the year of a good mixed diet, high in protein content and offering an abundance of vitamins, is most important.

Transfusion of blood and blood components is an effective, single measure for the control of persistent hemophilic hemorrhage. The recommended amount is 500 cc. of fresh blood or plasma, and repeated transfusions of 100 cc. of blood or plasma.

Thrombin may be used topically in the form of a dry powder directly upon the bleeding area. Other hemostatics beside thromboplastin are snake venom and placental extracts which have been used locally with some success. Care must be taken as to oral hygiene and the patient should be under the care of a dentist from early childhood.

*A preparation of oxalic and malonic acids.

It seems to be the consensus of opinion among a number of authors that thromboplastins given parenterally are of little value, and some authors such as Gold[4] feel that it is more or less therapeutic quackery to use them. Likewise such substances as vitamin K or the sex hormones have not been proven to be of definite value. The efficiency of oxalic acid or dicarboxylic acid has yet to be properly confirmed. The use of placental extracts, by mouth in young children, sometimes seems to have a marked effect, but occasionally it has been found that the clotting time is markedly prolonged by their use when given orally.

Tagnon[5] experimented with trypsin, given intravenously, on blood coagulation in hemophilia. He found that the action of intravenous injections of trypsin is of a short duration, less than one hour, and discontinued its use because its therapeutic effectiveness in hemophilia does not compare with transfusions of either fresh blood or plasma. Moreover, the toxicity of the drug is very great and it is dangerous to use.

Idiopathic thrombopenic purpura is the type in which splenectomy gives the best results. However, other measures should be tried first, such as the use of calcium chloride, histamine desensitization, para-thor-mone, and vitamin K.

The treatment of familial purpura consists of the following: correction of the anemia by blood transfusions, and absolute rest. Splenectomy is not of value in this condition.

SUMMARY

A short discussion of the more common types of purpura that may be encountered in the otolaryngologist's practice has been presented, also their laboratory and clinical findings. Some of the more recent developments and the treatment of these conditions have been brought forward. Two cases which the author prefers to call familial purpura have been cited.

CONCLUSIONS

The diagnosis of any one of the purpuras is not always an easy one. Farrar and Roxby[2] make this statement: "There are many typical cases of purpura that will not fit into either classification. In brief, the hemorrhagic manifestations in purpura often come from capillary dysfunctions, platelet deficiencies, and abnormalities."

The two cases that have been cited in this paper are proof of the above quotation. The author feels that the cases reported in this paper belong in the familial group.

A summary of the general treatment is as follows:

1. Proper diet is important.

2. Good hygiene and rest seem to form the basic therapeutic principle in all types of purpura.

3. Blood transfusions, preferably repeated smaller ones, seem to find a place in all cases of purpura.

4. Splenectomy gives the most dramatic result in idiopathic thrombopenic purpura.

5. Topical applications of various thromboplastins seem to be of more value than the oral administration or the hypodermic administration of any of the so-called drug coagulants now on the market.

However, constant research is going on as to the method by which blood coagulates. As more and more scientific truth about this phenomenon is discovered and dove-tailed with what is already known, it is hoped that some day there will be found a drug or substance as specific as insulin is in diabetes.

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X

SOME PRESENT-DAY CONCEPTS OF HEADACHE

AN ANALYSIS OF 1,000 CASES OF HEADACHE

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Headache in medical practice is a common complaint. Grimes[1] estimated that ten per cent of the patients that were seen in private practice complained of headache, whereas Weiss and English[2] claimed that 50 per cent of all patients have it as their chief complaint. Bassoe[3] stated that the disease is the commonest of civilized people, while Crile[4] maintained that it is a product of civilization. Petersen[5] regarded it as an expression of instability which is governed to a great degree by environmental factors.

Since the problem is so frequent, the question of what we know about it and how we cope with it is important. Proetz[6] stated that it is difficult for the student to obtain a balanced viewpoint from the literature on headache because many authors are victims and their interpretations are tinged with their own experiences. However, important contributions that are strictly objective and scientifically well controlled have been published since Proetz made this assertion. In the light of these facts, presented by Hilding,[7] Horton,[8] Williams[9] and Wolff[10] among many others, is it possible for us to regard the problem in a more specific manner? How much has the work of these men helped us in both the diagnosis and treatment of such complaints?

In order to answer these questions, 1,000 patients whose chief complaint was headache and who were referred for consultation to the Eye, Ear, Nose and Throat Department of the Camp Stoneman Station Hospital were analyzed. These patients were drawn from the station complement and from troops staging for overseas shipment. The anxiety attendant upon awaiting embarkation to combat zones was naturally great and was an important contributing factor in many cases. The patients were usually referred from their area dispensaries with the presumptive diagnosis of sinusitis, migraine or refractive error. A small number of patients were suspected of being malingerers and these presented an especially difficult problem, particularly when no objective findings were discernible.

A careful history was taken from all of the patients to determine the following facts about their complaints: the nature, onset, duration, frequency, site, extension, associated manifestations, aggravating factors, improving factors, family history and results of previous treatment. The physical examination included surveys of the eyes, ears, nose, throat, cervical spine; and, when indicated, laboratory tests, roentgenograms and medical, orthopedic, psychiatric and allergic consultations.

Eight hundred thirty-four patients had primary conditions as the basis of the complaint. One hundred fifty-two were acute cases and 682 were chronic. An arbitrary separation of acute from chronic was selected, eight weeks being the dividing line. In 362 instances only the primary condition was found, but in 472 remaining cases, secondary conditions were found which were either contributory to the headache or were secondary to it or to the conditions which produced it. In 166 cases that completed the series of 1000, the findings were such that either one of two or more factors or their combinations could be the basis for the complaints. Of these 11 were acute and 155 were chronic. Table 1 summarizes these figures.

TABLE 1.

	ACUTE	CHRONIC	TOTAL
Primary Diagnosis	152	682	834
Multiple Diagnosis	11	155	166

The generic diagnoses made in these cases are listed in Table 2. It is interesting to note that the same conditions are listed as primary, secondary, and accessory factors. (This is not contradictory; rather it is the result of the attempt to evaluate the findings in an endeavor to ascribe the major portion of the difficulties to a specific condition.)

Emphasis was placed on the effort to determine the prime etiologic factor since it was felt that only in this way could a scheme of treatment be decided upon and prompt and effective results in relieving the patient obtained. A proper accounting of all the possible factors contributing to the condition was equally important. Good results could hardly be expected if only one of several causative factors was treated. This was especially true where a self-perpetuating vicious cycle had been established. Relief in these cases was possible only by interfering with the mechanism responsible for such a cycle.

TABLE 2.

GENERIC DIAGNOSES ESTABLISHED IN 1,000 CASES OF HEADACHE.

DIAGNOSIS	PRIME FACTOR	ACCESSORY FACTOR	MULTIPLE FACTORS
Vasomotor Rhinitis	282	130	140
Myositis Cervicalis	241	208	181
Mechanical Obstruction of the Nose	90	84	100
Sinusitis	88	9	20
Neuropsychiatric Disturbances	75	10	56
Vascular Cephalagias	23	0	0
Ophthalmic Disturbances	11	6	18
Pharyngeal Involvements	5	13	34
Dental Disturbances	3	3	7
Cervical Spine Disturbances	9	0	0
Otitic Disturbances	2	9	6
Atrophic Rhinitis	4	0	0
Hypochromic Anemia	1	0	0

Vasomotor Rhinitis. The diagnosis of vasomotor rhinitis was made 537 times. In 282 instances it was the primary factor. Table 3 groups the cases in this class according to etiology.

Allergy was the commonest causative factor and was of both the intrinsic and extrinsic types. Based on a history of production of symptoms by chilling, exposure to sunlight, effort, or fatigue, 111 cases were classified as examples of intrinsic allergies. Although many cases of perennial rhinitis were caused by intrinsic and extrinsic factors, they were listed in this study as extrinsic allergies only. Eosinophile counts were not found to be of any assistance in differentiating the types.

The frequent association of the rhinitis with myalgia pointed out by Williams[9] was noted in this series. It was not, however, con-

TABLE 3.

VASOMOTOR RHINITIS

	ALLERGIC	MECHANICAL	INFECTIOUS	ENDOCRINE	PSYCHOGENIC	TOTAL
Primary Factor	231	1	48	2	0	282
Accessory Factor (Secondary & Multiple)	119	74	42	0	20	255

fined to those cases due to physical allergy alone. Four hundred seventy cases were found associated with myalgia of the head and neck.

The mechanical types of vasomotor rhinitis were all found to be secondary to septal deformities which produced pressure against the lateral nasal walls. Only one case was seen of external nasal deformity where it was felt that variations in the direction of the air currents was responsible for the irritation.

The infectious types of rhinitis were acute coryzas in 82 patients and specific bacterial sensitivities in five patients. The latter showed positive skin reaction to bacterial suspensions and were relieved by desensitization with specific vaccines.

The two patients with endocrine disturbances were females, one undergoing menopausal changes and the other in her fifth month of gestation.

Psychogenic vasomotor rhinitis was in no instance thought to be the primary basis for the headache. In 15 cases it was designated as the accessory factor and in five it was thought to be in the group of multiple causes.

For an explanation of the mechanism of headache resulting from vasomotor rhinitis we turn to Sluder's theory for the production of the vacuum headache.[11] This presumed that when a sinus was cut off from the outside air by blocking of the ostium, the entrapped air was absorbed by the blood producing a vacuum. Although this concept was generally accepted it was always subjected to much debate because of the inconsistency of the concept and the known facts about the physiological action of gases.

Accepted data on the rates of diffusion of gases and their tissue tensions indicate that there should be an initial increase in pressure

in a body cavity that is cut off from the outside air. This was never demonstrated in the paranasal sinuses but has been clearly shown to be true by Coryllos and Birnbaum[12] in the pleural space in artificial pneumothorax.

However, Hilding's decisive experiments have proved the soundness of Sluder's theory. They demonstrated the production of as much as -66 millimeters of water pressure in a dog's frontal sinuses as the cilia attempted to extrude a mucous plug through the nasofrontal duct.

Bearing this work in mind, it is easy to understand how negative pressure may develop in any of the sinuses in the presence of a vasomotor reaction which produces edema of the mucosa (thereby narrowing the ostia) and stimulates the secretion of large quantities of viscid mucus.

McAuliffe et al[13] showed that variations in intrasinusal pressure would produce pain. Gradual variations in pressure, either negative or positive, produced experimentally in the antrum caused pain. These workers showed that the areas most sensitive to pain were around the sinus ostia and that the pain was referred from the various intrasinusal and intranasal regions to specific points about the head and face.

Because of the work outlined above, it may be regarded as an accepted fact that any condition which will increase the turgor of the nasal mucosa, increase the mucoid secretion and diminish the size of the various ostia will invariably produce headache, if ciliary action is normal.

Regardless of the etiology of the vasomotor rhinitis, the clinical picture and the tissue response are found to be the same. Frequently, the etiology may be complex, so that the patient will present a picture of mechanical irritation due to a large vomerine spur, a family and situational history of allergy and psychosomatic influences. In such patients, when one or the other or all three factors are operating, the reaction observed in the nose will be unchanged.

Of the 282 cases in which vasomotor rhinitis was the primary condition, 157 cases were treated and followed up. Thirty-seven (23.5 per cent) were cured, 103 (65 per cent) were greatly improved and 17 (12 per cent) showed no improvement. Of course, the infectious group showed the best results—79 per cent cures. The patients in the allergic group were given skin tests, had submucosal sclerosing of their turbinates and were given decongestants.

TABLE 4.

MYOSITIS CERVICALIS

TYPES		PRIMARY	ACCESSORY	MULTIPLE
Trapezius Bilateral	Acute	59	37	15
	Chronic	83	132	83
Unilateral	Acute	31	8	4
	Chronic	25	28	38
Temporalis	Acute	1	0	5
	Chronic	2	2	16
Trapezius & Temporalis	Acute	9	0	0
	Chronic	17	0	0
Sternomastoid	Acute	8	0	0
	Chronic	6	1	2

Four and five-tenths per cent were cured and 80.3 per cent were markedly improved.

Myositis Cervicalis. Myositis cervicalis was diagnosed 635 times and 241 times it was the only condition to which the patient's complaints could be ascribed. In 208 instances it was seen as an accessory or secondary factor, while in 181 cases it was thought to be one of several factors that were equally responsible for the patient's complaints.

Myositis cervicalis has been written about extensively, yet it is a very neglected and relatively infrequently recognized condition. It has been described as rheumatic headache,[14] the cervical somatic syndrome,[15] indurative or myalgic headache,[16, 17] cervical fibromyositis.[18] It is so common a condition that Cabot in his differential diagnosis refers to it as the most frequent form of headache, Edinger[19] stated that two-fifths of all headaches were of this variety, while Auerbach[20] estimated that one-fifth to one-fourth of all headaches were of this nature. Despite these observations it has been the experience of the author that most medical men are unfamiliar with the condition, are unaware of its frequency and even regard the diagnosis with skepticism.

That the condition is not a newly described entity may be attested to by the fact that in the eighteenth century both Schönaich[21] and Morgan[22] wrote about it. Another description appeared in 1835 in a treatise on headache by Weatherhead.[23] Fifty years later Norström[24] published a pamphlet reporting a series of 36 cases of chronic head-

ache which he attributed to inflammatory deposits in the cervical muscles. Eleven years later he published another pamphlet in New York, entitled "Cephalalgia and Massage." From that time on there have been recurring reports. Thus, from Norström's last report in 1896 to 1914, four reports appeared discussing the condition and its treatment.[25-28] No further mention was noted until the 1923 edition of Osler and McCrae's[17] "Principles and Practice of Medicine" was published. This bore a brief description and introduced the term "rheumatic headache." Since then, 15 additional notes have appeared in the literature starting with Glasscheib's dissertation in 1927 and ending with Williams description of the intrinsic allergy syndrome in 1944.[10, 14-16, 18, 29-38]

Most of the early studies were chiefly clinical and speculative in character. Presumably this has been the reason the entity suffered by neglect in concept, diagnosis and treatment. The failure to evaluate and recognize a common condition has been a boon to some of the individuals beyond the pale of legitimate medicine who by their manipulations inadvertently gave patients relief from headache which the medical practitioner had not been able to offer to them.

A first assault in a scientific manner was made on the problem in 1922 by Schmidt[39] who excised the indurated nodules, sectioned them and failed to demonstrate any pathological changes. In 1938 Kellgren[32] plotted the distribution of pain referred to the head from injections of 4% sodium chloride solution into the posterior and lateral cervical muscles and the temporal muscles. Wolff and his coworkers[10] did a series of detailed and well-controlled experiments that aided greatly in understanding the condition and its mechanism. The facts gleaned from their work may be briefly outlined as follows.

Headache, regardless of its source, causes a reflex increase in tone in the cervical and temporal muscles. The muscles are affected in a definite sequence—first the nuchal ends of the trapezii are involved, then after a lag, the temporalis muscles increase their tension and after another relatively long lag the frontalis and occipitalis muscles are involved. When the ache is long continued, the muscles develop spastic nodules which are very tender and are themselves sources of pain. In corroboration of Kellgren's earlier observations it was shown that noxious stimuli applied directly to the cervical muscles produce headache and reflexly cause contraction of the unstimulated muscles. Thus, it was demonstrated that a vicious cycle, whereby headache produces spasm, which in turn becomes painful and produces further headache, could be reproduced experimentally. The occurrence of

increased muscle tone and subsequently spasm was also demonstrated in individuals in whom anxiety states were experimentally induced.

The actual physiology of this type of reaction has been described in detail by Lorent de No's[40] studies and analysis of the activity of chains of internuncial neurons. He stated that prolonged bombardment of pain impulses sets up a vicious circle of reflexes spreading through a pool of many neuron connections upward, downward, and even across the spinal cord. Because of the summation principle of nervous impulses, there is kept alive in such pools a constant circling of activity across the synapses involved. Thus, involvement of the sympathetic motor neurons in the lateral horns, which control vasomotor tone, produces spasm in the arteriole and venule ends of the capillary loops, raises the filtration pressure to cause edema and swelling. Cyanosis and anoxemia increase capillary permeability and filtration, further augmenting edema. Other synapses involved may be the anterior motor horn cells giving rise to skeletal muscle cramp and spasm.

To date, the one gap that exists in the facts compiled in support of the concept of the mechanism of headache and its relationship to the myositides is the experimental production of the increased tone and of spasm by "physical agents, as light, heat, cold. This has been observed clinically by most workers but no one has actually subjected a patient with a diagnosis of intrinsic allergy to controlled observations and electromyographic studies as the conditions of the experiment are varied. When these relatively simple observations are made, I believe that it will be the last link in the chain of facts necessary to prove the actual existence as a clinical entity of indurative myositis of the cervical and temporal muscles.

It was noted above that indurative myositis was found associated with vasomotor rhinitis in 470 instances. Because of the frequency of the association of these difficulties, Williams[9] thought that they were manifestations of the same basic disturbance. Seydell[16] and Freund[36] before him both termed myositis a manifestation of a physical allergy. However, in view of the work of Wolff and his group, I believe that myositis may be associated with the vasomotor rhinitis even when it is not on a physical basis.

As noted in Table 4, the condition was acute in 177 cases. These were treated with heat and massage and the results were usually very satisfactory and prompt. Eighty-five acute cases, with the myositis as the primary factor, were followed to completion and, of these, 84 per cent were cured. In a group of 60 chronic cases the

results were cure or marked improvement in 57. In the latter group, in addition to physical therapeutic measures, desensitization with histamine and infiltration of the skin and nodules with 0.5% procaine were used. It was not possible to judge the effects of treatment on this condition alone when it was either an accessory or secondary factor, for failures to clear the primary disturbances resulted in recurrences of the myositis.

Regardless of etiology, the presence of myositis in any case of headache must be recognized, since, as explained above, it may be a factor in the production of a vicious cycle of headache. As a result, treatment directed against a primary condition may be thought to be unsuccessful because of the persistence of the ache when the myositis is overlooked. As an example of just such a combination of factors, Case 611 is briefly recounted: O. N., a 23-year-old, white soldier complained of right frontal and occipital headache which occurred daily for four years. The ache was dull in nature and was worse in the early morning and at nightfall. Examination revealed a right inferior turbinate that was hypertrophied, a large right vomer spur and markedly tender nodules in the nuchal end of the right trapezius muscle. Palpation of the last caused the patient to complain of right frontal headache. After a submucous resection of the nasal septum, the headache persisted; when the myositis was treated, the nodules gradually cleared and the headaches disappeared completely.

The nasal architectural abnormalities in this case, together with the superimposed vasomotor responses, were responsible for increased intranasal pressure and resultant pain. The pain was greatest at times of greatest vasomotor response, i.e., early morning and at nightfall, when temperature variations and body chilling were most marked. This pain resulted in reflex trapezius spasm which when long continued became nodular in nature and a source of pain in itself. When the original basis of the pain was removed, the headache continued because of the nodules in the muscles and cure was obtained only when the cycle of pain-spasm-pain in the muscle was disrupted by physiotherapy.

Mechanical Obstruction of the Nose. Two hundred seventy-four patients with mechanical obstruction of the nasal fossae were seen. Twenty-five of these suffered from nasal polyposis and 249 had deviations of the nasal septum. Ninety cases were thought to have the obstruction as the primary basis, 84 were regarded as being due to accessory factors and 100 cases were classified as due to multiple factors. The deviations were separated into three anatomical types:

TABLE 5.

MECHANICAL NASAL OBSTRUCTION

CONDITION	PRIMARY	ACCESSORY	MULTIPLE
Polyposis	3	8	14
Vomerine spurs	28	2	14
Dislocations	29	65	69
Impactions	30	9	3

vomerine spurs great enough to produce pressure on the lateral nasal wall, dislocations of the septal cartilages causing obstruction to both breathing space and sinus drainage, and finally impactions against the turbinates without obstruction of the airway.

The production of headache by definite abnormalities in the nasal architecture is hardly a debatable matter, particularly when the consideration of a superimposed vasomotor action is added. Spurs become pressure points when the mucosal turgor increases and polypi or septal obstructions cause a damming back of the mucosal stream which flows more or less continuously through the sinus ostia. The latter phenomenon may result in the formation of a mucous plug which in turn may be responsible for a diminished intrasinus pressure.

After plotting the points of reference of pain from the various nasal and paranasal structures, McAuliffe[13] concluded that if a zygomatic frontal, temporal or vertex pain is not greatly reduced in intensity or eliminated by shrinkage or local anesthetization of the nasal structures, it is in all probability not due to disease of these structures. This is an important point to bear in mind in evaluating the abnormalities uncovered in the nose, providing the role of muscular reflex spasm in contributing to the headache is also considered.

Forty-eight cases in the primary group were followed up after treatment and only one patient failed to derive benefit from surgical correction of the abnormality. Many cases like the one cited above (Case 611) required additional treatment for the secondary factors. The question may be posed at this point as to the necessity of this additional treatment and the possibility of the cycle of muscular spasm and pain running itself down after the generating mechanism has been abolished. That this will happen there is no doubt, for patients have submitted to surgery and then have not been seen again for reobservation or treatment for a year or more. On reexamination, they

TABLE 6.

TYPES OF SINUSITIS

INVOLVEMENT		PRIMARY	ACCESSORY	MULTIPLE
Pansinusitis	Acute	11	0	3
Unilateral	Chronic	11	1	6
Bilateral	Acute	4	0	0
	Chronic	2	2	0
Frontal	Acute	7	5	2
	Chronic	1	0	0
Ethmoid	Acute	2	0	2
	Chronic	4	0	3
Sphenoid	Acute	0	0	0
	Chronic	3	0	0
Maxillary	Acute	24	0	0
	Chronic	19	1	5

have been found to be symptom free. The actual benefits of the operation were long delayed for each patient stated that symptoms persisted for months after the operation.

Sinusitis. Sinusitis was one of the commonest of the presumptive diagnoses in the referred cases. It was substantiated, however, in only 118 instances and these were classified as shown in Table 6.

Of the 88 patients whose sinus infections were judged to be the prime basis for their headaches, 49 had acute involvements. Eighty-three of these patients were followed to completion of treatment and only one, a patient with maxillary sinusitis, failed to obtain relief.

The failure to designate a sinusitis as the primary basis of headache may be questioned. This may be explained by citing a specific case (Case 184). This patient complained of recurrent right frontal headache. Examination revealed a suppurative right frontal sinusitis, a right trapezius myositis, a vasomotor rhinitis and a large cellular right middle turbinate bone. Any one of these factors alone could produce the headache, hence it was not reasonable to select one and designate it as the primary cause and ascribe secondary roles to the others. This is especially true in a case of sinusitis which is secondary to conditions which may themselves cause headache.

Hypothesizing a sequence of events leading to the patient's complaints one may presume that the hypertrophied turbinate plus the vasomotor rhinitis produced mechanical obstruction of the nasofrontal duct which lead to the infection. The pressure variations, with and

TABLE 7.

NEUROPSYCHIATRIC CONDITIONS.

DIAGNOSIS	PRIMARY	ACCESSORY	MULTIPLE
Old Skull Fracture	4	0	1
Post-Traumatic Syndrome	12	1	32
Anxiety States	26	3	13
Hypochondriasis	19	0	2
Constitutional Psychopath	4	1	0
Reactive Depression	3	0	0
Schizophrenia	0	0	1
Moron	0	0	1
Neuralgias—Supraorbital	4	0	0
Infraorbital	2	1	3
Sphenopalatine	1	4	2

without infection, in the frontal sinus produced headaches which resulted in trapezius muscle spasm. Treatment directed at establishing frontal sinus drainage by crushing the cellular turbinate, irrigating the frontal sinus and reducing the mucosal swelling as well as the use of heat and massage on the trapezius muscle brought about prompt relief. Mere treatment of the sinusitis would not have dissipated the headache as quickly.

Neuropsychiatric Disturbances. Seventy-five cases were diagnosed as neurologic or psychiatric disturbances. The commonest condition, as would be expected, were the anxiety states. This was understandable in precombat troops at the port of embarkation. Sick calls, which at most stations amounted to five per cent of the personnel, jumped to 20 per cent. Awareness of the psychosomatic aspects of headache permitted the true nature of most of the cases to be recognized and only the occasional extreme cases were sent for consultation. The presence of these conditions as accessory and multiple factors was common as noted in Table 7.

The post-traumatic headaches represented a large group of 45 cases that posed difficult diagnostic problems. Most of the individuals had no visible sequelae of their head injuries. Five showed evidence of old skull fracture roentgenographically and were listed separately. The symptoms following the trauma were in no way related to the type or degree of injury. All the cases of this type in the series had associated myositis cervicalis.

That actual pain may result from an anxiety state was definitely demonstrated by Wolff and his group as noted above in the discussion on muscle spasm. A patient suffering from an anxiety neurosis and a reactive depression was submitted to study with electromyographic tracings of his cervical muscles. When some of the basis for the patient's alarm reactions was removed, the tone of the muscles was shown to be definitely diminished. Depriving the patient of his security immediately returned the myographic readings to their former levels and caused a recurrence of the headache. From this one may presume that the psychosomatic headache was produced by these variations in muscular tone and spasm.

The mechanism of the post-traumatic headache has been investigated many times and the most favored theories are: 1.) increased intracranial pressure; 2.) dilatations of the ventricular system; 3.) stretching of duro-arachnoid adhesions; 4.) vasomotor instability; 5.) dilations of the arteries in the scalp and the dura; 6.) muscular spasm. Friedman and Brenner[41] studied the relationship of the post-traumatic syndrome to the histamine headache produced experimentally to determine if possible the mechanism of the former. They injected histamine intravenously into patients with post-traumatic headaches and discovered that it usually reproduced their headaches. From this they concluded that the vascular sensitivity resulted from the trauma, that the arteries were probably toneless and relaxed so that the increase in blood pressure distended them and caused pain or that the change in caliber of the vessels produced the pain.

Ross and McNaughton[42] studied a group of patients with chronic post-traumatic headaches employing electro-encephalograms and Rorschach disability ratings. They found that the patients with localized aches showed less evidence of cerebral damage than the others. Those with bizarre and generalized aches had previous personality instabilities similar to the psychoneurotics, which showed up clearly in the Rorschach tests.

The patients in this group responded poorly to all types of treatment. The various physiotherapeutic measures usually caused the patients to complain of an increase in their difficulties. Long series of treatments with histamine diphosphate were similarly unsuccessful. Infiltrations of the indurated areas in the muscles brought about only transient relief. As a result, not one of the patients followed up was found to be helped in any way.

TABLE 8.

OPHTHALMIC DISTURBANCES

CONDITION	PRIMARY	ACCESSORY	MULTIPLE
Refractive Errors	10	2	8
Phorias	1	0	4
Amblyopia Ex Anopsia	0	1	5
Anterior Synechia	0	0	1
Optic Atrophy	0	1	0
Old Retinitis	0	1	0
Ptosis	0	1	0

The neuralgias encountered responded readily to treatment. The acute cases were treated with heat, vitamin B_1 and vitamin B complex. Three of the supraorbital neuralgias and four of the infra-orbital neuralgias were chronic cases and were controlled completely by alcohol blocks. Most of the cases of sphenopalatine neuralgia were secondary to intranasal disturbances and management of these brought prompt relief.

Ophthalmic Disturbances. When headaches are recurrent and associated with use of the eyes, it is common to ascribe them to some ophthalmic disorder. Approximately one-third of the patients referred to the clinic came with a presumptive diagnosis of a refractive error. This was far in excess of the findings. Actually only 35 patients were found to have eye disorders. Of these, 11 were primary causes and 24 were secondary or accessory factors (Table 8).

Eckardt, McLean and Goodell[43] demonstrated experimentally that noxious stimuli in various parts of the eye will produce headache. Increases in intra-ocular pressure, traction on the extra-ocular and intraocular muscles, as well as eye strain produced artificially by lenses, could produce aching, eye discomfort, heaviness of the head and headache.

In evaluating ophthalmic disturbances in order to judge their role in contributing to the production of headache it is important to keep in mind the observation made by Cushman[44] that refractive errors present for many years are unlikely to exert a malign influence unless other factors are added, such as overuse of the eyes, debili-

TABLE 9.

VASCULAR CEPHALALGIAS.

Histamine Cephalalgia	5
Migraine	13
Hypertension	3
Aneurysm of the Circle of Willis	1

tating illness or psychic disturbances. Thus, aches of recent origin should be due to recent causes.

Vascular Cephalalgias. The various headaches that are thought to be due to disturbances of the intracranial vascular structures are grouped under this heading. These may be vasomotor, hypertensive, arteriosclerotic or aneurysmic in nature. In the age groups dealt with in this study, the hypertensive and arteriosclerotic types were not common. Only one case of aneurysm of the circle of Willis was encountered. Although migraine was a very common presumptive diagnosis for patients referred for consultation, only 20 cases of vasomotor headaches were found. This disproportion was probably the result of the tendency to label any severe headache associated with nausea and vomiting and not having an obvious basis as "migraine."

Migraine is thought of as a definite symptom complex entailing headache that is sudden in onset, persistent in nature and associated with nausea and vomiting. However, the term should be reserved only for those periodic headaches resulting from vascular disturbances, associated with aura, lasting more than 12 hours and relieved by gynergen. All headaches associated with nausea and vomiting, vertigo, blurring of the vision, are not migraine headaches, since it has been shown that these symptoms may be produced by vasomotor nasal disturbances, by cervical myalgia[10], by eye strain, by ear disturbances and even by cervical vertebral arthritis.[45, 46]

The vasomotor disturbances may be either vasodilations, vasospasms or a combination of both. The histamine headache so clearly recalled by Horton[47] is vasodilatative in nature and has been referred to as erythromelalgia or "red" headache. It is made worse by histamine and improved by epinephrine or strong pressure over the external carotid artery or its temporal branches. The vasodilatative nature of the condition is manifested by injection of the conjunctiva, plugging of the nose, epiphora, and flushing of the face. The pain is sudden in onset, short in duration (less than one hour) and occurs

TABLE 10.

PHARYNGEAL DISTURBANCES.

DIAGNOSIS		PRIMARY	ACCESSORY	MULTIPLE
Pharyngitis	Acute	0	0	2
	Chronic	3	3	3
Adenoiditis		0	1	3
Thornwaldt's Disease		2	0	0
Tonsillitis	Acute	0	1	4
	Chronic	0	8	22

mostly at night after going to sleep. The distribution of the pain conforms to the ramifications of the external carotid artery.

The migraine victim usually has an allergic background and is overly reactive to emotion. He is highly intelligent, restless, very conscientious and above average in ability and drive.[48] Van Storch[49] showed that these patients are more sensitive to histamine than are those who rarely or never have headaches.

The actual vascular reaction in migraine occurs in two stages. First, there is a vasoconstriction which is responsible for the aura, scotomata, and the cortical phenomena. During this phase, the patient is usually pale and the ophthalmic aura when present consists of white flashes; hence this may be referred to as the "white" headache. Gynergen administered at this stage will usually abort the attack.

Then follows a vasodilatation permitting increased pulsations and stretching of the vessels at the base of the brain.[49]

Studies of the physiological mechanism of migraine by Pfeiffer et al[51] led to the conclusion that the excursions of the blood vessels could be traced to blood volume changes (hemoconcentration) unassociated with compensatory changes in peripheral vascular tone. Accordingly, treatment should aim at increasing the blood volume or restoring an intense vascular tone.

Histamine desensitization as recommended by Horton proved to be very successful in the cases of histamine headaches that were treated in this series.

Poor control of the migraine patients and inability to follow them up prevented the evaluation of any mode of treatment.

Pharyngeal Disturbances. The conditions affecting the pharynx and associated with headaches are listed in Table 10.

The association of headache and a pharyngeal disease may be readily explained anatomically[52] since pathways may be traced along the sensory branches of the sphenopalatine ganglion to neurones and synapses leading back to the pain center of the trigeminus in the post-central gyrus. In only five of the 52 cases listed was it possible to ascribe the headaches primarily to the pharyngeal condition.

All of the patients in this group had associated indurative myositis of the trapezius muscles. This may have been due to the pharyngeal condition directly by involvement of the prevertebral space, as explained by Proetz,[53] or may have been secondary to the headache itself as a reflex reaction.

The incidence of headaches and Thornwaldt's disease has been noted many times. Yankauer,[54] in reporting observations on 155 cases, noted that headache was a presenting symptom in 68 per cent. Godwin[55] reported some cases and described a characteristic headache syndrome. In the cases noted in this series, removal of the bursa and treatment of the secondary myositis resulted in complete relief.

TABLE 11.

DENTAL DISTURBANCES.

DIAGNOSIS	PRIMARY	ACCESSORY	MULTIPLE
Impacted Molar	1	0	0
Abscessed Molar	1	2	0
Costen's Syndrome	1	1	7

The commonest dental disturbance noted in this group was vertical malocclusion producing a temperomandibular disturbance that has been described by Costen.[56, 57] In a study of 400 such cases Costen found that headache was a presenting complaint in 107 instances.

The condition should be suspected whenever there are missing molars, poorly fitting dentures or marked overbite and both ophthalmological and rhinological causes are ruled out.

Spinal Disturbances. Eight patients were found with definite osteo-arthritic changes in the cervical spine and one patient with an extruded nucleus pulposus. All the patients were afflicted with marked cervical myositis and fibrositis. Scant attention has been directed to these conditions as a cause of headache as shown by the few references in the literature.[45, 46]

TABLE 12.

OTOLOGIC CONDITIONS.

DIAGNOSIS	PRIMARY	ACCESSORY	MULTIPLE
Acute External Otitis	2	0	1
Eustachian Salpingitis	0	5	0
Traumatic Otitis	0	3	2
Otitis Media	0	0	3

The patients treated for this condition in this series were not helped by the facilities available (foreign protein injections and short wave diathermy).

SUMMARY

A review of 1,000 cases of headache revealed that the commonest causes in the order of frequency were: vasomotor rhinitis, myositis cervicalis, mechanical nasal obstruction, paranasal sinusitis and neuropsychiatric disorders. Less frequent causes were the vascular cephalalgias, ophthalmic, pharyngeal, dental and cervical spinal disturbances.

Vasomotor rhinitis was diagnosed 552 times but could be designated as the prime cause of headache in only 282 instances.

Myositis cervicalis was found 630 times and was regarded as the prime cause in 241 instances. In 470 patients, vasomotor rhinitis and myositis cervicalis were associated. Despite this frequency, the two may or may not be attributed to the same etiological factor. Thus they may both be manifestations of a physical allergy, but an understanding of the mechanism of the reactions of the cervical muscles indicates that the myositis may be a secondary reflex from a headache caused by the vasomotor rhinitis.

The importance of recognizing the presence of an indurative myositis and the vicious cycle of headaches for which it may be responsible has been pointed out. It has been indicated that this cycle may be responsible for the continuation of a headache long after the prime cause has been corrected.

Mechanical nasal obstructions were discovered in 274 patients. In 90, they were believed to be primarily responsible for the headaches when combined with the normal vasomotor variations of the nasal mucosa.

Paranasal sinusitis did not prove to be as common a cause of headache as presumptive diagnosis would indicate. Its existence was substantiated in only 117 patients and the results of treatment were excellent.

Neuropsychiatric disorders were uncovered in 141 cases. Anxiety states and post-traumatic syndromes predominated. Treatment was unsatisfactory.

Ophthalmic disturbances were discovered in 35 cases and were primary causative factors in 11 patients.

Although the vascular cephalalgias were presumptive diagnoses in fully 33 per cent of the referred cases, confirmation was made in only 22 patients. Migraine is a definite symptom complex and the term should not be applied to any headache because it is associated with nausea and vomiting.

CONCLUSIONS

The patient with a headache must be studied in an orderly manner which will evaluate all the factors that may either directly cause, increase or prolong the ache. An understanding of the mechanisms by which the various abnormalities discovered in the course of the physical examination may produce headache, as well as a knowledge of the neuro-anatomic pathways for referred pain is essential. Recent researches have resulted in clearer concepts of the mechanics of the vacuum headache, spastic muscular headache, vascular cephalalgia and psychosomatic headache. The important role of the spastic cervical muscle in the production of or the prolongation of headache has been noted again. Evaluation of the possible causative factors based on these concepts permits the planning of effective schemes of treatment.

57 WEST 57TH STREET.

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XI

CONGENITAL CHOANAL ATRESIA. A NEW SURGICAL APPROACH

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Atresia of the choana is a rare congenital anomaly and was first reported by Otto in 1829. In an exhaustive survey of the literature in 1945 Boyd[1] found 230 reported cases. The atresia occurs at the junction of the hard and soft palates and extends to the base of the sphenoid. About 90 per cent are bony atresias and 10 per cent are membranous. Kazanjian[2] believes that a fair percentage are of the mixed type.

Although choanal atresia is now generally accepted to be congenitally acquired, there seems to be a low incidence of associated congenital anomalies and of hereditary predisposition. The embryology can only be described as obscure. Existing theories are (Fig. 1):

1. Persistence of the upper part of the buccal (or buccopharyngeal) membrane which normally ruptures very early, at about the 3 mm. stage.[3] The septum and medial-growing palatine processes of the definitive choanae fuse with this membrane.

2. Persistence of the nasobuccal membrane which normally ruptures in the sixth week, at about the 10 mm. stage.[4]

3. Overgrowth of the palatine processes at the choanae so that they fuse not only with the septum but also with the base of the sphenoid.[5]

These hypotheses are all very difficult either to prove or disprove. The most popular conception seems to be the persistence of the nasobuccal membrane. One disadvantage of this theory is that

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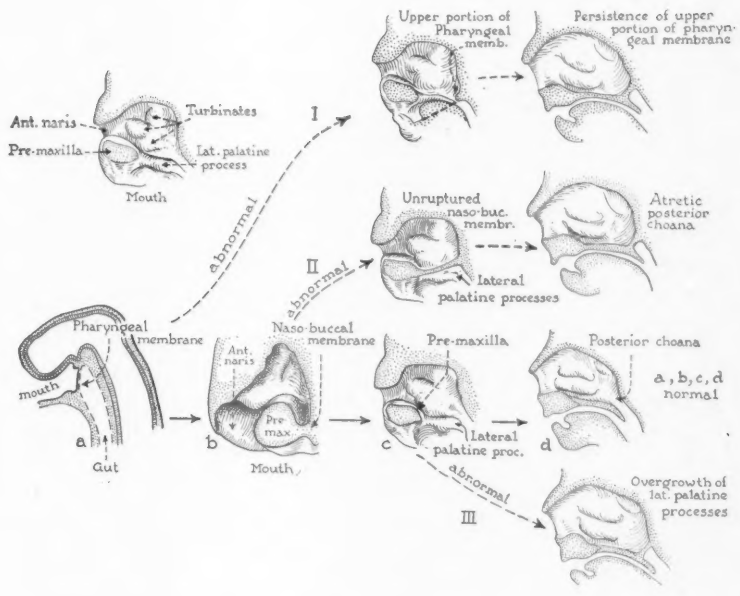

Fig. 1.—Diagram of the embryological development of the choanae.

Figs. a, b, c, d represent normal development (after Arey[6]).

Figs. I, II, III represent deviations from the normal resulting in atresia of the choana as described in the accompanying article.

(a) 2.5-mm stage before rupture of the pharyngeal membrane.

(b) 12-mm. stage before rupture of the nasobuccal membrane to form the primitive choana.

(c) 7-week stage after rupture of the nasobuccal membrane. The primitive choanae are still present but the lateral palatine processes are growing medially to divide the nasal cavity from the mouth and thus form the definitive choanae as seen in (d).

(d) Developmental stage at full term.

the nasobuccal membrane originally extends between the sphenoid and the premaxilla, and it can be argued that the atresia would have to extend to the incisive foramen, which it does not do. The atresia is bony or membranous depending upon how much mesoderm remains between the ectoderm of the nose and that of the mouth.

REPORT OF A CASE

The patient is a 23-year-old white female who came to the Northwestern Otolaryngology Clinic complaining of a mucopurulent discharge from her left nostril of three years' duration. Posterior rhinoscopy revealed no choana on the left side. The patient was unable to blow her left nostril. When questioned it appeared she had never been able to do so. A probe introduced through the left anterior naris was prevented from passing into the nasopharynx by what appeared to be a bony obstruction posteriorly. Mercurochrome and later lipiodol introduced into the left nostril did not reach the nasopharynx. An x-ray film with lipiodol showed the presence of an atresia of the choana which was thicker below than above.

There was no familial history of similar anomalies or of deaths in newborn brothers and sisters. (Bilateral choanal atresia is thought to be a possible cause of suffocation in the newborn.[8, 9]) She had never had any ear trouble and her ears and hearing were normal. She had a highly arched hard palate. (These signs are found in most of the cases in the literature.) There had been no previous nasal surgery or adenoidectomy though such operations have often been performed in these cases.

On closer examination it was noted that the lateral nasal wall on the atretic side protruded medially at its posterior aspect and that the floor of this nostril became elevated so that posteriorly it was about one centimeter above the floor of the other side. This finding had previously been reported by Kazanjian[2] in one of his cases. Other features not previously mentioned were a lateral deviation of the posterior portion of the nasal septum toward the affected side and a medial displacement of the lateral wall of the nasopharynx on the obstructed side so that the left eustachian tube orifice lay almost in the midline. Lipiodol displacement showed absence of the left sphenoid with a partial pneumatization of the left side by the right sphenoid. Typical glairy mucus on the atretic side has been described in the literature. In this case the secretions were thick, white, and opalescent. Nasal smears revealed many leucocytes but no eosinophiles.

The literature describes many diverse procedures for the relief of this condition by the use of chisels, gouges, curettes, cautery, burrs, and electrocoagulation by either of two routes: splitting the soft and hard palates or via the anterior nares. The nasal septum route has frequently been used. It appears that the most important step in preventing closure is the removal of the posterior portion of

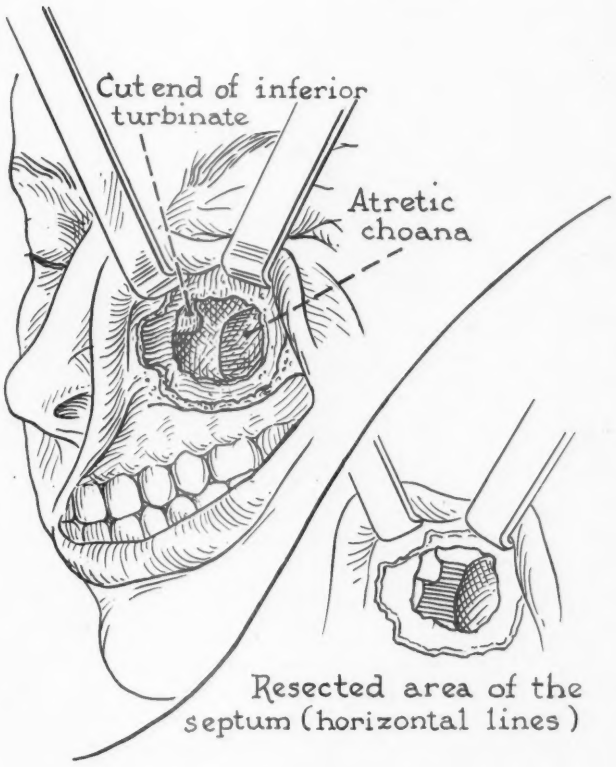

Fig. 2.—Diagram of the transantral approach to the choana. After a Caldwell-Luc incision, the antrum in entered through the canine fossa. A second window is made into the nose in the posterior portion of the maxillary sinus, and the posterior tip of the inferior turbinate is removed. The resected area of the septum is shown by horizontal lines.

the nasal septum. Kazanjian[2] added an important adjunct to the intranasal exposure of the operative field by incising and retracting laterally the columella and the cartilaginous septum.

Adequate exposure of the operative field seemed to be an even more than usually important consideration to the surgical treatment of this case because of the unusual medial displacement of the lateral wall of the nose and nasopharynx, including the sphéno-palatine ganglion and the internal maxillary artery with its large branches

to the nasal cavity. One of the authors suggested that an even better exposure might be made by a Caldwell-Luc incision with removal of the anterior face of the antrum and by making a second window through the medial wall of the antrum at its posterior aspect. According to Dr. Elmer Hagens this procedure had an added advantage in providing ready access to the sphenopalatine artery via the posterior wall of the antrum in case of unexpected and uncontrollable hemorrhage. Dissections were carried out on the cadaver and the above-described method with resection of the posterior tip of the inferior turbinate achieved better exposure than any of the methods described in the literature.

The patient was admitted to Wesley Memorial Hospital and the operation was performed after blocking the second division of the trigeminal nerve via the posterior palatine canal. This canal was normally located and easily accessible, and the anesthesia was complete for one and one-half hours without use of other topical anesthesia. The usual Caldwell-Luc procedure was carried out revealing a normal maxillary sinus. On removing the lateral wall of the nose into the inferior meatus a thick, hard, bony ledge was found projecting medially at a level with the posterior border of the antrum. Just before it made contact with the septum there was a shallow blind trough. When the posterior portion of the nasal septum was resected it was found that the thick bony ledge projected medially only slightly more than the lateral wall of the nasopharynx. It was decided that resection of this ledge might open into the sphenopalatine fossa and at best would produce a source of much granulation tissue. Therefore it was not disturbed. A large rubber tube was inserted through the anterior naris and passed through the perforation in the septum into the nasopharynx. The Caldwell-Luc incision was closed in the usual manner.

The postoperative course was uneventful. The rubber tube was removed after 48 hours and the patient discharged from the hospital. No packs were used and there was no bleeding. She was followed in the clinic and advised to snuff physiologic sodium chloride solution through her nose. Epithelialization of the margin of the septal perforation was almost complete in one week and the opening in the septum has remained the same size (about 1.5 cm.) to date (6 months). The patient breathes through both sides of her nose. Slightly more air passes through the right (normal) than the left (operated) nostril, but she can blow either side equally well and is very well satisfied. Mucoid discharge from the left side of the nose

continues to be greater than from the normal side, but since she can now blow her nose it causes very little inconvenience.

In chronic infection and allergic conditions ciliary epithelium is sometimes largely replaced by goblet cells. It was considered that such a change might have occurred secondary to the long standing choanal obstruction and that faulty ciliary activity might be responsible for this accumulation of mucus, which normally should be disposed of via the posterior nares. A study of ciliary function was made by distributing lampblack over the nasal mucosa. Both sides of the nose were swept clean equally well in apparently normal time (15 minutes). As the execessive nasal secretion from the front of the nose has been decreasing progressively since the operation, it is regretted that the cilia were not checked earlier.

CONCLUSION

A case of congenital atresia of the choana is reported with a new transantral approach.

We believe that:

1. Local block anesthesia via the posterior palatine canal is the anesthesia of choice in adults.

2. That the most important procedure in the correction of unilateral atresia of the choana is the resection of the posterior portion of the nasal septum.

3. Minimum manipulation and absence of packs and other foreign material postoperatively appeared to be responsible for the minimum infection and granulation seen during healing. In many of the cases described in the literature postoperative packs, cautery, insertion of tubes over a long period of time, and even epidermal skin grafts on the mucosal surfaces were employed because of the tendency toward cicatricial contraction of the new opening.

The advantages of the transantral approach are:

1. Better exposure of the operative field.

2. Ready control of hemorrhage, if it occurs, by ligation of the internal maxillary artery.[7]

3. Less danger of injuring the pterygopalatine nerves and blood vessels.

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XII

THE USE OF THROMBIN IN NASAL SURGERY

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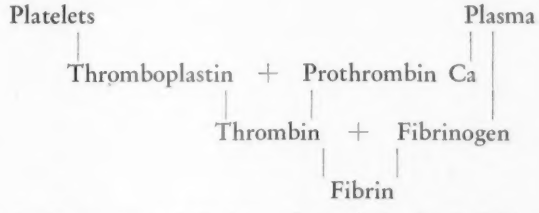
A preliminary report was previously published on the use of thrombin in a series of 34 nasal operations.[1] At that time the author concluded that thrombin is a valuable adjunct in rhinologic surgery. To date, 152 nasal operations, including submucous resection, or combined submucous resection and rhinoplasty, have been performed in which thrombin was employed. Stevenson[2] previously reported a small series of cases in which thrombin was employed in submucous resection or intranasal antrostomy with excellent results.

Thrombin is not only an effective hemostatic agent for the control of capillary bleeding, but also serves as an excellent tissue "glue" in promoting rapid adhesion of wound edges and tissues. These properties are valuable in nasal surgery in lessening tissue reaction, in preventing hematoma (especially of the septum), in reducing postoperative bleeding, in effecting a more rapid restoration of the nasal airway, and, in rhinoplasty, in shortening the period of postoperative edema.

According to Quick,[3] only three agents are essential for the coagulation of blood: fibrinogen, prothrombin Ca and thromboplastin. The first two are supplied by the plasma in amounts far in excess of the requirements for effective coagulation of blood. The third, thromboplastin, is not free in the plasma but is probably locked up in the platelets and only becomes available when the latter disintegrate. As long as the platelets remain intact, therefore, the blood remains fluid. The clotting process can be expressed diagrammatically as follows:



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When a vessel is cut, there is a deposition of platelets about the torn edges of the vessel, which form an adherent mass from which needles of fibrin arise and radiate into the lumen. This fibrin reticulum enmeshes the cellular constituents of the blood and thus forms the clot. Intact platelets attach themselves to the fibrin shafts and by a mechanism that is poorly understood cause the clot to retract, narrowing the lumen and pulling in the torn edges, thus securing a firm fibrin mass to occlude the vessel. The laying down of fibrin serves only a temporary purpose, as fibrin is not a permanent structural material and is either absorbed or replaced by connective tissue.

From this brief description it can be seen that the application of pure thrombin to cut tissues will effect an immediate deposition of fibrin by the interaction of the thrombin with the fibrinogen naturally present in the tissue plasma. Since no intermediate products need come into play and since thrombin is one of the two final interacting products in this reaction, hemostasis can be effected by the use of thrombin even in true hemophilia and other bleeding tendencies, with the rare exception of afibrinogenemia.[4]

In skin grafting and wound suturing, thrombin applied topically serves a double function: it effects prompt hemostasis, and the precipitated fibrin anchors the graft in place or "glues" the wound edges together. These are valuable adjuncts in plastic surgery and many reports have been published expounding the virtues of thrombin in this work.[5-9] By its use, postoperative hematoma and infection have been virtually eliminated, the use of compression bandages has been dispensed with, and the convalescent period has been materially shortened.

Application and Technique. To date 152 nasal operations have been performed in which thrombin was employed as a hemostatic and tissue-fixative. Of these, 110 have been submucous resections of the nasal septum, 25 have been rhinoplasties, and 17 have been combined submucous resections and rhinoplasties. In addition, thrombin has been employed in a number of cases of radical sinus surgery and in numerous other operative procedures on the ear, nose and throat.

All of the nasal operations were performed under local anesthesia, using one dram of a freshly prepared 5% solution of Alypin in 1:2000 epinephrine solution as a topical spray, supplemented by submucosal injections of 1% procaine hydrochloride solution. The patients previously had received barbiturate and morphine-atropine sedation. At the time of operation a thrombin solution is prepared

by dissolving the powdered contents of one ampule of thrombin (5000 Iowa units) in 5 cc. of sterile, isotonic sodium chloride solution, thus making a solution containing 1000 units of thrombin per cc. This is placed in a sterile 5 cc. syringe, to which is attached a two-inch dull-pointed fistula needle.

After completion of the surgical procedures during submucous resection and before insertion of the packing, all free blood and clots are removed from the nose and especially from between the septal flaps by careful spot-suctioning. Approximately 3 to 4 cc. of the thrombin solution is then sprayed between the septal flaps, and the flaps are held in apposition for fully 3 to 4 minutes by the use of a long-bladed nasal speculum of the Carter type. (The author has modified this speculum by flattening the blades and eliminating the concavity in them, thus allowing better contact between the septal flaps). The flaps will then be found tightly adherent. It is the author's custom to place two No. 0000 black silk sutures in the septal incision, though this is not essential. A drop of the thrombin solution is carefully placed over the incision and at the site of any mucosal tear that may have occurred during the operation. If any turbinal or intranasal sinus surgery has been performed, thrombin solution is likewise sprayed over the cut surfaces after removing all free and clotted blood with suction. The nose is then packed. For this purpose, the author prefers two 3/4 inch "cigarette drain" packs on each side, supplemented by ½-inch iodoform or petrolatum gauze tape for the ethmoid and vestibular regions. This packing is believed to provide better splinting for the septum and is removed very easily and with considerably less discomfort and bleeding than gauze alone. The packing is usually removed in from 15 to 24 hours.

In rhinoplasty, after completion of the operation, the thrombin solution is sprayed beneath the skin of the dorsum and firm pressure is applied over the nose with gauze for several minutes. Bleeding ceases almost immediately and the skin becomes tightly adherent to the underlying bone and cartilage. It is essential to place the skin carefully and exactly where desired, as after a few minutes it can be moved only by the use of a knife or periosteal elevator to break up the fibrin adhesions. If a submucous resection was performed at the same time as the rhinoplasty, the usual packing is inserted after spraying thrombin solution between the septal flaps as described above. The packing is then left in place for 48 hours. If only a rhinoplasty has been performed, Oxycel packing is inserted in the vestibule only and this is removed after 48 hours. A properly made aluminum splint, lined with felt, is carefully applied and left in place

for from three to five days, depending upon whether osteotomies were performed.

Comment. In practically every case where thrombin was applied topically to cut surfaces of the nose, a mild stinging sensation was experienced by the patient. No untoward reactions were observed in any case, even in those in which some of the thrombin solution was swallowed, and no instance of allergic reaction was observed.

Stasis of capillary bleeding was practically immediate, but in the cases in which a bone artery had been severed, thrombin was ineffective in maintaining hemostasis.

The septal flaps became tightly adherent within a few minutes and remained so thereafter. The incision became tightly sealed in every case, requiring re-incision in the two cases in the series in which hematoma occurred.

It was striking that the septum remained quite thin and tight following the application of thrombin, so that in from three to five days there was a clear airway in practically every case and all were free of the usual septal bogginess.

Only two septal hematomas resulted in this series. In one case, a bone artery was cut in the removal of the anterior nasal spine. This was not properly dealt with at the time of operation, and within 30 minutes after the removal of the packing, the septal flaps were bulging and in contact with the lateral walls of the nose. A large quantity of fresh blood was evacuated from the septum by re-opening the incision, the edges of which were tightly "glued" and required dissection by a fine-pointed hemostat after removing one of the sutures. Nasal packing was tightly inserted as the bleeding into the septum continued after the removal of the hematoma. The patient made a delayed but good recovery.

In the second case, the patient was operated on at the request of an allergist, as she was showing no improvement to intensive desensitization therapy. The septal mucosa was boggy and bled readily throughout the operation. At the time of the removal of the packing, considerable bleeding was encountered and this persisted for some time. The next day an anterior septal hematoma was recognized and evacuated by reopening the incision, but this refilled almost as fast as the blood could be evacuated, and it was felt that

a bone vessel was at fault. Packing was reinserted after instilling thrombin and penicillin solution between the septal flaps, and no further trouble was encountered. The patient showed considerable edema of the nasal tissues postoperatively and developed several adhesions that subsequently required excision. This was attributed to the second packing and the state of allergy that existed. The ultimate result, though delayed several weeks, was good from an anatomical standpoint.

With the exception of the above noted cases, there was no instance of postoperative hemorrhage. The house staff members and the nursing staff have volunteered the observation that the patients on whom thrombin was employed had less postoperative oozing through the packing, and that much less bleeding occurred when the packs were removed. In this connection it might be said that at one time we were completely out of thrombin for several days, during which time one submucous resection was performed. It was the universal observation of every one associated with the operation or the after-care of the patient, that in this case there was more bleeding postoperatively and convalescence was considerably delayed as compared to the cases in which thrombin was employed.

In one case an abscess of the septum occurred in a man with secondary atrophic rhinitis on the wide side of the nose. However, there was no evidence of hematoma or abnormal bleeding from the nose, and the abscess did not appear until the seventh postoperative day. This was evacuated by incision, and a mixture of thrombin and penicillin was instilled into the abscess cavity preparatory to repacking of the nose. No further treatment was needed and the packing was removed in 48 hours.

In the cases of rhinoplasty, it was observed that the skin over the dorsum became tightly adherent within a matter of a few minutes and there was considerably less postoperative edema and almost no ecchymosis. There was no case of postoperative infection. In every case the edema had subsided and the nose was practically normal in appearance in from two to three weeks. It was possible to discard the nasal splint in three to four days in cases in which osteotomy had not been performed, and in four to seven days in cases in which osteotomy had been performed. (Previously the splint was worn for at least 10 days and frequently for 14 or more days, depending upon the degree of edema present and whether an osteotomy had been performed.)

CONCLUSIONS

1. There is presented a series of 152 cases of nasal surgery in which thrombin was employed topically as a hemostatic and tissue-fixative. In 110 cases submucuous resection of the nasal septum was performed, in 25 rhinoplasty alone, and in 17 cases combined submucous resection and rhinoplasty was performed.

2. The topical use of thrombin in nasal surgery is effective in producing and maintaining stasis of capillary bleeding, but it is not effective against active bleeding from cut vessels.

3. In submucous resection of the nasal septum, the use of thrombin within the septal flaps, as described, results in the flaps becoming tightly adherent within a few minutes and remaining so thereafter, so that in from three to five days there is a clear airway in practically every case. Postoperative oozing was reduced to a minimum, there was no case of postoperative hemorrhage, and only two cases of septal hematoma resulted in the entire series. (In both instances the hematoma resulted immediately upon the removal of the nasal packing and was attributed to a cut bone vessel.)

4. In rhinoplasty, the topical use of thrombin produces immediate fibrin-fixation of the tissues and aids materially in preventing postoperative edema, ecchymosis and thickening of the nasal tissues.

5. The author considers thrombin a valuable adjunct in nasal surgery.

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XIII

ACUTE MASTOIDITIS MASKED BY PENICILLIN THERAPY

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The treatment of acute otitic infections was radically changed by the advent of sulfonamides. After the early reports, it appeared that acute mastoiditis was destined to be among the rare diseases. It was not long before failures were observed and certain organisms were found to develop a resistance to the drug. The literature now contains many accounts of the masking effect of sulfonamides in the treatment of acute otitis media and its complications. The introduction of penicillin was then hailed as the answer to all temporal bone infections in which the sulfonamides had failed. Since Florey and Florey[1] first reported the use of penicillin in otitic infection, there have been numerous accounts of the miraculous results of the drug in curing acute infections of the middle ear and mastoid. There is no doubt that the discovery of penicillin has created a new conception of the treatment of acute osteomyelitis. As long as the drug was difficult to obtain and was used only in carefully selected and supervised cases, the results were most encouraging. Since penicillin has been available to all, it has, in some cases, been used indiscreetly, inadequately and unwisely. Now some of the same difficulties are being encountered as were observed in sulfonamide therapy. Very few cases of mastoiditis are seen by the otolaryngologist until some type of treatment with sulfonamides or penicillin has failed. The drugs are often given in small doses without bacterial diagnosis, and the familiar signs and symptoms of acute mastoiditis are altered.

Information concerning the chemistry of penicillin is meager, except that Abraham and Chain[2] report that it is an unstable organic acid. For clinical administration the more stable sodium and calcium salts are employed. Early studies[1, 3, 4] of the pharmacologic properties of penicillin showed that its antibacterial action was greatest when administered intravenously or intramuscularly. It can be given subcutaneously but this method is more painful and more

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irregular in action. The drug is very effective when used locally but is rather ineffective when given orally or by rectal instillation. Cooke and Goldring[5] have shown that the distribution of penicillin in the body is by the vascular route. It is distributed uniformly through the tissues with a good blood supply but it diffuses poorly into the pleural cavity and is seldom detected in the cerebrospinal fluid. Spink[6] states that penicillin is usually bacteriostatic in its action but, under certain circumstances, the action can be considered to be bactericidal or bacteriolytic. In patients with normal renal function about 60 per cent of the amount injected is excreted in the first hour. Since its action is one of bacteriostasis, requiring contact with the bacteria for an appreciable length of time, frequent injections of the drug are necessary. There is no satisfactory chemical method for determining penicillin levels in the blood or the urine. Penicillin is effective against a number of organisms, the most frequent in otitic infections being the streptococci, pneumococci and staphylococci. There are a number of organisms not susceptible to penicillin, but very few[7, 8] investigators have reported bacteria becoming penicillin resistant.

The usual pathologic condition of acute mastoiditis should be considered. The causative bacteria are variable but hemolytic streptococcus is by far the most frequent organism. Pneumococci, especially type III, nonhemolytic streptococci, and staphylococci are responsible for most of the other otitic infections. All of these organisms, except for an occasional strain, are susceptible to penicillin and the incidence of other types of mastoid infection is small.

Essentially, mastoiditis is an inflammation of the lining of the mastoid cells, beginning with hyperemia and followed by edema and exudation. It occurs as a complication of acute otitis media and the virulence of the invading organism determines the spread of the pathologic process. The lining of the mastoid cell is actually mucoperiosteum which is adherent to the underlying bone. As the edema develops, the lining becomes very thickened, almost filling the cell. This is followed by a serous effusion which fills the bony cavity. The vessels of the mucoperiosteum are markedly engorged and later become thrombotic. Leucocytic infiltration follows and the exudate becomes purulent. The infection extends through the mucoperiosteum and involves the bony trabeculae of the mastoid cells. Infected granulations replace the destroyed epithelium and bone. The pressure exerted by the purulent material and the insufficient blood supply produce additional necrosis. In the phlegmonous form of mastoiditis, inflammation and infiltration are in the

foreground and suppuration is secondary. As vascular engorgement, venous thrombosis and round cell infiltration progress, suppuration, osseous necrosis and abscess formation occur, and the coalescent form of mastoiditis is produced. The acute process may follow one of several courses. In favorable cases, it may subside or the granulations may become organized and be followed by regrowth of bone. The osteomyelitis may progress, producing cavitation and abscess formation, or it may extend to vital structures resulting in complications.

In the coalescent form of acute mastoiditis, the blood supply is impaired and, as penicillin is distributed by the blood, its action is not uniform throughout the infected area. A mottled pathological process is encountered similar to that observed by Galloway[9] and Babcock[10] in the treatment of mastoiditis with sulfonamides. The areas with a good blood supply show healing with healthy granulation tissue and new bone formation. Deep in the areas of healing where the tissue is relatively avascular are found softening, abscess formation and extensive necrosis. The tympanic membrane may appear almost normal and the middle ear infection apparently heals, but the infection continues and is progressive in the mastoid. Cody[11] described a similar condition as erysipelatous mastoiditis in which he compares the pathologic process to that of erysipelas of the skin. This smoldering process seems to have an affinity for the sinus and dural plates and is frequently complicated by meningitis and lateral sinus thrombosis.

Treatment of acute mastoiditis with penicillin usually gives prompt and often dramatic clinical response. In a limited number of cases it produces a confused clinical picture. This depends upon the infecting organism, the amount of the drug administered, and the progress of the pathological process when treatment is instituted. The virulence of the infection is rapidly reduced unless the causative organism is insensitive to penicillin. Putney[12] considered the bacteria to be penicillin resistant if there was no clinical improvement in three or four days of treatment.

The patients usually show marked improvement in 48 hours and the pain disappears unless some complication such as meningitis or extradural abscess is present. When the infection is retarded but not cured, there is frequently a dull pain or sensation of heaviness on the affected side. The pain and the sensation of fullness are similar to that seen in streptococcus mucosus capsulatus infections of the middle ear and mastoid.

The temperature usually drops to normal within a short time after penicillin is started but irregular elevations follow if the mastoid

infection is not controlled. A sudden rise of temperature generally indicates the onset of some complication, but with the use of penicillin the temperature curve is not characteristic. Occasionally a patient develops a sensitivity to the drug and has a marked elevation of temperature due to this. There is usually an accompanying skin rash which distinguishes it as an allergic reaction.

The amount of aural discharge is variable. In the phlegmonous form of acute mastoiditis, the discharge stops promptly but recurs when the drug is discontinued if there is any residual infection. In most cases, the discharge is absent or is markedly reduced. In some instances, as long as the penicillin is given, the discharge is changed to a thick mucoid drainage but becomes purulent again when the drug is discontinued.

The tympanic membrane is seldom seen bulging in the posterior superior quadrant but remains inflamed and thickened if infection persists in the mastoid. Perforations fail to heal. In two cases the thickened tympanic membrane was retracted against the medial wall of the middle ear and there was no drainage after myringotomy. The characteristic sagging of the posterior superior canal wall is usually absent. The impairment of hearing depends upon the amount of middle ear involvement. In general there is less hearing loss than in mastoid infections not treated by penicillin.

Tenderness over the mastoid is almost always absent. This is usually indicative of a thin cortex and of infection in the region of the mastoid antrum. These cells overlying the antrum have a good blood supply and infection in the region tends to clear up when treated with penicillin. In one case a subperiosteal abscess developed during penicillin therapy.

The leucocyte count drops as the virulence of the infection is reduced but will rise rapidly when the drug is discontinued if the mastoid infection is still present. Penicillin does not affect the hematopoietic system as do sulfonamides.

The findings in roentgen examination of the mastoid may be altered by penicillin therapy. Acute mastoiditis in a pneumatic bone has definite x-ray characteristics. Administration of penicillin causes the cell content to appear more translucent and the bony structure to be more distinct than in untreated cases. In one patient a second x-ray film showed improvement in the mastoid infection but the clinical symptoms persisted. At operation the superficial cells under

the cortex were almost normal but extensive necrosis was found in the sinus plate and the bone covering the posterior fossae. The roentgenologist should be advised that the drug has been used.

Most of the complications of mastoiditis are masked by penicillin. In lateral sinus phlebitis the characteristic fluctuating temperature is altered. The rise is not as great as in untreated cases and the chill at the height of the temperature is usually absent. A positive blood culture cannot be obtained if the patient has received several injections of the drug before the blood is collected. Two cases of lateral sinus thrombosis developed while the patients were receiving penicillin therapy, and in neither did the drug control the disease until the sinus was opened and the thrombus removed. After operation the response to treatment was rapid. Ligation of the jugular vein has been considered unnecessary when the patient is receiving penicillin but in both of these the vein was ligated. Meningitis does not produce as high a temperature when treated with penicillin. To control the infection, the drug is injected intrathecally as well as intramuscularly, since it does not diffuse into the cerebrospinal fluid in appreciable amounts. Adhesive arachnoiditis has been reported as a complication of intrathecal administration. In two patients treated, the temperature remained elevated and the meningitis was not controlled until the infection in the mastoid was removed at operation. After operation both responded promptly to treatment. Koebbe and Potter[13] advocate a combination of the sulfonamide drugs and penicillin in the treatment of otitic meningitis. Smith[14] reports a case of petrositis which showed improvement after mastoidectomy and the administration of penicillin but the patient continued to have relapses until the petrous pyramid was drained.

CONCLUSIONS

Penicillin will cure most cases of acute otitis media if given in adequate doses, the exceptions being the ones due to bacteria that are insensitive to the drug. In the phlegmonous form of acute mastoiditis penicillin is very effective. After the infection has progressed to the stage of coalescent mastoiditis the drug alone is not sufficient. The severity of the disease process is reduced but the usual clinical symptoms and findings are often distorted. Adequate surgical intervention is necessary when bone destruction is extensive and when complications are present. Penicillin is an efficient agent in sterilizing the bloodstream, controlling the spread of infection, and is a beneficial supplement to surgical treatment.

SUMMARY

1. Indiscreet, inadequate and unwise administration of the drug is responsible for most failures of penicillin in the treatment of acute mastoiditis.

2. Penicillin is distributed to the tissues of the body by the blood. It is bacteriostatic in its action but is eliminated rapidly by the kidneys.

3. In coalescent mastoiditis the blood supply to the infected area is reduced and the distribution of penicillin is not uniform. A mottled pathological process results.

4. The response to penicillin therapy depends upon the infecting organism, the amount of the drug administered, and the progress of the pathological process when treatment is instituted.

5. The signs and symptoms of acute mastoiditis may be altered by treatment with penicillin.

6. Penicillin therapy changes the clinical picture of lateral sinus phlebitis, meningitis and petrositis.

7. Surgical intervention in addition to the administration of penicillin is necessary in the majority of such cases to effect a cure.

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XIV

SEVERE EPISTAXIS AND ITS MANAGEMENT: REPORT OF ELEVEN CASES IN WHICH THE EXTERNAL CAROTID ARTERY WAS LIGATED

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The term "severe epistaxis" as considered here is applied only to nasal hemorrhage requiring hospitalization for its management. Numerous cases in which minor nosebleed was adequately controlled in the offices of the Mayo Clinic or in the emergency rooms of the hospitals connected with the clinic are not included in this review.

Since 1930, severe epistaxis has been treated in hospitals connected with the clinic in 106 cases. These are classified as to source of bleeding and cause whenever these data could be determined. The methods of control that were used are described, including a report of eleven cases of ligation of the external carotid artery.

A knowledge of the anatomy of the blood supply of the nose is of practical advantage in deciding which method to employ in treating the hemorrhage and in determining possible length of hospitalization and possibility of recurrence. The arterial blood supply of the interior of the nose is derived from both the internal and the external carotid arteries. The first branch of the internal carotid (the ophthalmic artery) and branches of the external carotid artery (the internal and external maxillary arteries) participate in the blood supply. Only the principal branches are described here.

The sphenopalatine artery, the terminal branch of the internal maxillary artery, is the chief arterial supply to the nasal fossa and its appendages.[1] It reaches the nasal cavity through the sphenopalatine foramen, located on the lateral nasal wall near the posterior end of the superior nasal meatus. Immediately after entering the

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nasal fossa, the artery divides into a medial and a lateral branch. The lateral branch spreads forward over the turbinates, the meatuses, the maxillary and ethmoidal sinuses, and anastomoses with the posterior ethmoidal artery of the internal carotid system and with branches of the external maxillary artery of the external carotid system.

The medial branch of the sphenopalatine artery, called the nasopalatine artery, crosses the undersurface of the body of the sphenoidal sinus, enters the septal wall and spreads anteriorly and inferiorly over the septum.

The descending palatine artery arises from the internal maxillary artery, descends through the pterygopalatine canal, passes through the hard palate, enters the nasal cavity by way of the incisive canal and ends on the nasal septum. The pharyngeal artery, also a branch of the internal maxillary, goes to the roof of the pharynx and from here gives off branches to the posterior and superior portions of the nasal fossa and the sphenoidal sinuses.

The anterior and posterior ethmoidal arteries arise from the ophthalmic artery as it passes along the medial wall of the orbit. The anterior ethmoidal artery enters the nasal cavity along the side of the crista galli and spreads downward over the anterior superior part of the nasal cavity and the adjacent sinuses and over the anterior part of the septum.

The posterior ethmoidal artery enters the nasal cavity through an aperture in the cribriform plate, spreads over the lateral nasal wall, the posterior ethmoidal cells and the upper part of the septum, and anastomoses with branches of the sphenopalatine artery of the external carotid system.

The external maxillary artery (facial artery) supplies blood to the anterior portion of the septum through the septal branch of the superior labial artery.

There is a relatively free anastomosis between the adjacent branches of the external carotid artery of the same side and with the corresponding branches of the opposite side. Johnson and Foster[2] have estimated that collateral circulation is established within five days after ligation of the external carotid artery. The final distribution[3] of these vessels is found in the mucoperiosteum in three layers of capillary networks: one deep in the periosteum, a second surrounding the glands and a third layer just beneath the epithelium.

The paranasal sinuses receive an additional blood supply through the outer bony walls.

The determination of the causes of epistaxis is at best an inexact study. Of the various causes found in this review (Table1) only trauma to the nose, either accidental or surgical, is considered a direct cause of nasal hemorrhage. The other causes are not considered as being more than contributory to the incidence of epistaxis. Trauma figured in only eight per cent of the cases, while in nearly all the remaining cases the nosebleed began when the patient was asleep or at rest. Analysis of the age distribution (Table 2) is sufficient to show that severe epistaxis is a geriatric problem, the incidence increasing directly with the age. The most common causes encountered were those conditions which become progressively worse as the patient grows older. Of all the patients studied, approximately 40 per cent had either hypertension or arteriosclerosis or both, previously diagnosed. Forty per cent of all patients were more than 60 years of age. Of those past 60 years, 60 per cent had hypertension. In 29 cases, presenting no obvious cause of hemorrhage or previous diagnosis of cardiovascular disease, the epistaxis was classified as of undetermined cause. In nearly all of these, however, the patients were in the age group in which cardiovascular disease is common.

It is worthy of note that such conditions as neoplasm, "vicarious epistaxis," and alterations in the constituents of the blood played only a small part in the causation of nasal hemorrhage, although the depression of the level of the blood platelets due to excessive loss of blood may have caused a prolongation of the epistaxis.

Few textbooks and papers on epistaxis do more than mention the anterior portion of the septum as a source of serious nosebleed. Most dismiss it as being of only nuisance value, but of the patients whose cases are reviewed here, half of the total number requiring hospitalization bled from this source. It is not surprising to those who routinely have to treat nosebleed that anterior septal bleeding can be so severe as to require hospital apparatus for its control. Three out of five patients more than 60 years of age bled from this source.

Nearly a third of all patients (Table 3) bled from a point high and posterior in the nose, most of these from the sphenopalatine artery. Trauma incidental to packing the nose prior to hospitalization was found to be a considerable factor in those cases in which bleeding was from the turbinates or diffusely from the nasal membranes.

The severity of epistaxis may vary from a slight dripping of blood to a fatal hemorrhage and the treatment must be applied accordingly. Whatever the cause or source of the hemorrhage, the principal object in the treatment is to stop the flow of blood as soon as possible.

On admission to the hospital, the patient is examined first for symptoms of shock and for active bleeding. If the patient is an adult he is given 1/6 or 1/4 grain (0.01 or 0.016 gm.) of morphine by hypodermic injection. With close observation of the patient, it is not difficult to distinguish between shock due to hemorrhage and the shocklike appearance of a patient who is nauseated from swallowing too much blood. If there is uncertainty as to the condition of the patient, there need be no hesitancy in giving him a transfusion of blood. Liters of group 0 blood should be available for immediate use.

If the hemorrhage is seen to be controlled by packs inserted prior to hospitalization, the packs are left in place and the patient is watched thereafter for recurrence of bleeding.

If there is active bleeding, all packs are removed and the nose is quickly cleaned with a suction apparatus under direct vision to determine the source and the severity of bleeding. Cotton pledgets moistened in equal parts of 1:1,000 solution of epinephrine chloride and 10% solution of cocaine are then used to shrink the nasal mucous membrane and provide local anesthesia for any necessary instrumentation. A length of 1-inch (2.5-cm.) gauze heavily covered with vaselin is probably the most useful agent available to the rhinologist for the control of nasal hemorrhage. Large spurting vessels do not respond to chemical cautery, and high posterior spurting vessels are inaccessible to the electrocoagulating point. A short strip of vaselin gauze, carefully and firmly packed over the bleeding point, controlled hemorrhage in 80 of the cases of nosebleed reviewed here (Table 4).

There should be no hesitancy about inserting a postnasal pack if the anterior packing alone fails to stop the flow of blood. The posterior pack should be made large enough so that the choana is completely occluded when the pack is in place. The insertion of a posterior pack is a matter of a few seconds and causes the patient only momentary discomfort. The pack may be left in place for from 24 to 36 hours, although the hazards accompanying its use must be kept in mind; namely, otitis media and nasopharyngeal abscess. Blood was seen in the middle ear in eight cases, in one of which myringotomy

was required for the relief of earache. Chemoprophylaxis or antibiotics are routinely used in cases in which posterior packing is required.

A case of osteomyelitis of the basisphenoid with meningitis and cranial nerve palsies resulting from a nasopharyngeal abscess following the use of a nasopharyngeal pack was treated at the clinic recently and is being reported in another paper.[4]

Topic application of thrombin and thromboplastin, even strips of salt pork are useful in diffuse bleeding from the mucous membrane but are of little value in stopping a spurting type of hemorrhage.

Eleven patients failed to respond to conservative treatment and required ligation of the external carotid artery. Two of these developed minor wound infections and a third patient needed a tracheotomy because of laryngeal edema secondary to wound infection.

Five deaths occurred among the cases reviewed. One was attributed to coronary occlusion, another was of a two-and-a-half-year old infant with disease of the blood-forming organs. The other deaths were due, respectively, to hemorrhage from esophageal varices, pulmonary hemorrhage secondary to bronchiectasis, and overwhelming toxicity secondary to scarlet fever.

Ligation of a principal artery for the control of hemorrhage from one of its branches was first performed more than 150 years ago. According to Bartlett and McKittrick,[5] who reviewed all cases of ligation of the carotid artery up to 1917, Abernethy[6] attempted, without success, to tie off the common carotid artery for hemorrhage in 1795. The first successful ligation of the common carotid artery was reported by Flemming[7] in 1803. Since that time, the common carotid artery has been ligated hundreds of times for a variety of conditions, including nasal hemorrhage.

Ligation of the external carotid artery alone for nasal hemorrhage has been more limited. McKnight[8] in 1926 reviewed the literature and found 17 instances of ligation of the external carotid artery for nasal hemorrhage up to that time. All were successful. Of these, two were cases of spontaneous epistaxis, reported by Jackson.[9] McKnight did not mention the case reported by Hyde,[10] who successfully tied off the external carotid artery for idiopathic nasal hemorrhage in 1925. Abrahams,[11] in 1928, and Bricker,[12] in 1932, each reported a case of ligation of the external carotid artery for postoperative nasal hemorrhage. Hawthorne,[13] in 1930, and Johnson and Foster,[2] in 1933, each reported one case of ligation for traumatic nasal hemorrhage. Barker,[14] in 1943, reported two cases of ligation

of the external carotid artery for nasal hemorrhage due to trauma. Strauss,[15] in 1933, reported a case of ligation of the external carotid artery for severe spontaneous epistaxis in a woman in the fifth month of pregnancy. The epistaxis was successfully stopped but the patient aborted a four-and-a-half-month old fetus a few days later.

Goodyear,[16] in 1937, described a case in which he ligated the anterior ethmoidal artery for nasal hemorrhage. The ligation was performed through an incision along the eyebrow. Hirsch,[17] in 1936, described a case of ligation of the internal maxillary artery for nasal hemorrhage. The ligation was performed by means of a transmaxillary approach, originally described by Seiffert[18] in 1928.

The indication for ligating the external carotid artery becomes evident when the nasal hemorrhage persists or recurs with severity and the level of the hemoglobin and the erythrocytes drops steadily in spite of repeated nasal packing and transfusions of blood. It is not safe to wait until the patient has lost so much blood that a state of shock supervenes. Bean[19] in 1937 reported that in two of 300 cases of coronary occlusion, the occlusion was caused by the sudden fall of blood pressure accompanying hemorrhage, one of the cases being a case of spontaneous epistaxis. McKinlay[20] described a case of coronary insufficiency precipitated by hemorrhage from a duodenal ulcer. McLaughlin and others[21] reported a similar case in 1940.

Blumgart and others[22] found 11 out of 350 cases of coronary occlusion to be due to shock. They emphasized the importance of preventing a fall of blood pressure under any circumstance in persons who have coronary sclerosis.

Ligation of the external carotid artery has been performed at the Mayo Clinic in 11 cases of nasal hemorrhage since 1930 (Table 5). In each instance the bleeding was controlled. In two cases the epistaxis followed an operation on the sinuses. In the remaining cases, the epistaxis occurred spontaneously, although in four of the cases there was hypertension, previously diagnosed. In one case (Case 11, Table 5) in which the diagnosis of coronary sclerosis had been made several years previously, precordial pain and symptoms of shock developed at the time of admission. An electrocardiographic tracing showed evidence of occlusion of a coronary artery. The patient failed to respond to treatment adjusted to combat both the cardiac lesion and the state of shock. The left external carotid artery was ligated with the patient under local anesthesia and the hemorrhage was controlled. However, the patient died several hours later.

SUMMARY

Since 1930, 106 cases of severe epistaxis requiring hospitalization for its control have been treated at the Mayo Clinic. Half of the patient were 55 years of age or more. Forty per cent of the patients had hypertension or arteriosclerosis or both. In half of the cases the bleeding was from the anterior portion of the septum. In most of the cases nasal packing, alone or in conjunction with cautery, controlled the bleeding. In 11 cases ligation of the external carotid artery was necessary.

MAYO CLINIC.

TABLE 1.

CAUSES OF EPISTAXIS: ANALYSIS OF 106 CASES.

CAUSE	CASES	PER CENT
Undetermined	29	27
Hypertension and Arteriosclerosis	19	18
Hypertension without Arteriosclerosis	14	13
Arteriosclerosis without Hypertension	10	9
Trauma	9	8
Familial Hemorrhagic Telangiectasia	6	6
Postinfection	5	5
General Debility	5	5
Postoperation	4	4
Blood Dyscrasia	4	4
Malignant Lesion	1	1

TABLE 2.

AGE DISTRIBUTION.

AGE, YEARS	CASES	PER CENT
Less than 5	2	2
5-14	3	3
15-24	7	6
25-34	6	5
35-44	20	19
45-54	17	16
55-64	25	24
65 and over	26	25

TABLE 3.

SOURCE OF BLEEDING

SOURCE	CASES	PER CENT
Anterior Portion of the Septum	54	51
Sphenopalatine or Nasopalatine	20	19
Diffuse	14	13
Undetermined: High and Posterior	13	12
Turbinates	5	5

TABLE 4.

TREATMENT USED IN CONTROLLING EPISTAXIS

TREATMENT	CASES
Nasal packing	38
Nasal Packing and Cautery	23
Nasal Packing. Cautery and Transfusion	19
Chemical Cautery Only	7
Electrocoagulation Only	5
Submucous Resection	2
Injection of Foreign Protein	1
Transfusion and Ligation of External Carotid Artery	11

TABLE 5.

LIGATION OF THE EXTERNAL CAROTID ARTERY FOR NASAL HEMORRHAGE.

CASE	AGE, YRS.	CONTRIBUTORY FACTORS	SOURCE OF BLEEDING	HEMOGLOBIN GM. PER 100 CC. OF BLOOD	ERYTHROCYTES PER CU. MM. OF BLOOD
1	53	Sinus operation	Right sphenopalatine artery	8.2	2,700,000
2	60	Hypertension, group 3	Left sphenopalatine artery	7.0	2,000,000
3	31	Sinus operation	Right sphenopalatine artery	10.8	4,000,000
4	38		Left sphenopalatine artery	12.7	
5	50	Hypertension, group 2	Left sphenopalatine artery	6.7	3,900,000
6	54	Hypertension, group 3	Left sphenopalatine artery	10.8	4,000,000
7	51		Left nasopalatine artery	8.0	2,900,000
8	51		Right sphenopalatine artery		
9	61	Hypertension, group 4	Right sphenopalatine artery	7.0	2,500,000
10	62		Right sphenopalatine artery	6.4	1,800,000
11	70	Generalized arterio-sclerosis	Left sphenopalatine artery	10.0	3,500,000

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XV

THE TREATMENT OF CERTAIN SPECIFIC TYPES OF HEADACHE WITH HISTAMINE

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In the management of the numerous aches and pains which occur in the regions of the head and neck, a great variety of pathologic and nonpathologic conditions must be considered in diagnosis and treatment. Recent reports by Proetz[1] and by Horton[2] present very comprehensive classifications of these various conditions.

One of the most common types of headache encountered by the otolaryngologist is that with pain referred to the frontal, ocular and maxillary regions and in which no demonstrable pathological condition is found. These cases are usually classified as migraine, Sluder syndrome or atypical trigeminal neuralgia. This group constitutes a large percentage of those patients whose primary complaint is "sinus trouble." Other patients with sinus trouble who show demonstrable pathological conditions usually prove to have nasal allergy or suppurative sinusitis.

In 1939, MacLean, Craig and Horton[3] described a new syndrome of vascular headache designated as "erythromelalgia of the head." Later, in 1941, the term "histamine cephalgia" was adopted by Horton.[4] To Horton, we should be most highly indebted for he has presented the solution to one of the most common types of headache. More recently, Shea,[9] has reported his observations on the diagnosis and treatment of this type of headache.

Since February 1941, we have encountered a group of 126 patients whose primary complaint was headache referred to the region of the frontal sinus, the eye, the maxillary sinus and sometimes the temple, the mastoid and the occiput. Thirty-two of these patients had typical histaminic cephalgia. Although the remaining 94 had a similar type of pain, these were considered as an atypical group.

Of the 32 typical patients, 23 were men and 9 were women. The ages varied from 27 to 67 years and were distributed in decades

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as follows: third decade, 1, fourth decade, 12, fifth decade, 7, sixth decade, 10 and seventh decade, 1. The duration of symptoms ranged from six months to 28 years.

The syndrome of histaminic cephalgia reported by Horton[4] may be described as follows: It is characterized by a unilateral headache, effecting mostly individuals in the later decades of life, which is of short duration as it usually lasts less than an hour. It begins and often terminates suddenly, tends to awaken the patient at night one to two hours after going to sleep and is frequently eased by assuming the sitting position or standing erect. It is associated with a profuse lacrimation and congestion of the eye, rhinorrhea or stuffiness of the nose, increased surface temperature and often swelling of the temporal vessels of the involved side of the head.

The pain, described as constant, excruciating, burning, and boring, involves the frontal area, the eye, the temple, the face and the neck. The pain is not associated with the distribution of any cranial nerve, but conforms to the ramifications of the external carotid artery. During and following attacks the branches of the external and common carotid arteries may be sensitive to pressure. At the beginning of an attack compression of the common carotid artery and sometimes the temporal artery may give prompt relief.

The pain is almost always unilateral, rarely bilateral but may occasionally shift from one side to the other. The attacks are not associated with the menses.

Among the 32 typical cases 10 patients were subjected to some surgical procedure as follows: 3, dental extractions; 1, nasal cautery; 2, submucous resection; 1, extensive radical sinus surgery; 1, tonsillectomy; and 2, resection of the trigeminal nerve. Attacks of pain were precipitated by cold air in 5 patients, by sunlight in 1 and by alcoholic beverages in 6.

One of the characteristic features of histaminic cephalgia is the occurrence of attacks of pain at night. Although most of the patients have pain both at night and during the day, some may have it only at night. At night the attacks of pain come on suddenly and awaken the patient out of a sound sleep. The patient usually gets out of bed to assume the upright position because the pain becomes intolerable in the reclining position.

In 35 of Horton's[4] 72 patients typical attacks of unilateral pain were induced by the subcutaneous injection of 0.10 to 1.2 mg. of histamine. No attempt was made to induce attacks in the other

patients. By means of electric thermocouples, Horton showed that during an induced attack the surface temperature on the involved side rose more rapidly and to a higher level than that on the other side of the head. Induced attacks of this type could not be produced in normal individuals. In five patients Horton was able to relieve the pain in both induced and spontaneous attacks by the intravenous injection of a 1:400,000 solution of epinephrine. Placing the hands in ice water also immediately relieved the attacks of four additional patients.

REPORT OF TYPICAL CASES

CASE 1.—R. C., a man, aged 33, was first observed on February 6, 1941, complaining of attacks of pain referred to the left upper jaw, the eye and the frontal region. Four days previously he had had the left upper second molar tooth extracted. Nothing definite was found to account for the pain. When the patient returned again on February 28, 1941, he still complained of attacks of pain over the left frontal sinus, the eye and over the region the canine fossa. Further interrogation revealed that he had been having these attacks for the previous five years. Occasional attacks occurred on the right side. The pain was described as deep and boring and was accompanied by sweating of the skin over the left side of the forehead. During attacks the sweating continued even when exposed to very cold weather. The attacks would come on and end suddenly. They lasted from 30 or 40 minutes to six or seven hours. Attacks appeared at night and aroused him from a sound sleep.

Examination during an attack revealed sweating of the left frontal area, lacrimation, and bogginess of the nasal mucosa on the affected side. During a symptom-free interval, an attack of headache was induced by the injection of 0.15 mg. of histamine. Subsequent treatment, beginning with injections of 0.01 mg. and gradually increasing to 0.20 mg., relieved the attacks. Later smaller maintenance doses of 0.01 mg. were found to be satisfactory.

CASE 2.—J. M. H., a man, aged 47, reported for examination on January 18, 1944. On this day he had planned to enter a hospital to have a resection of the right trigeminal nerve for the relief of attacks of pain of 20 years' duration, referred to the region of the right frontal sinus, eye, and cheek. Attacks occasionally occurred also on the left side. For many years the attacks would come on about once a year lasting four to six weeks. When first observed the attacks had been present for six months. He had had no previous trouble for a period of six years. The pain was excruciating

in character. He complained of burning and tingling of the skin over the affected area. It was also sensitive to touch. There was sweating on the involved side, as well as marked lacrimation and watery drainage with stuffiness of the right side of the nose. Attacks would come on at night awakening him from sound sleep. The pain usually lasted from one to one and one-half hours. There were no trigger areas and the pain was not brought on by eating, shaving, or touching the face. Therefore, none of the characteristics of trigeminal neuralgia was present.

The examination of the ears, nose and sinuses was negative. During an attack, sweating was observed over the affected area and sensitiveness of the temporal vessels to pressure was demonstrable.

Treatment was started with the subcutaneous injection of histamine, using at first 0.10 to 0.20 cc. of a 1:1,000,000 dilution. This gave some temporary relief. The dosage was gradually increased until it was found that 0.40 to 0.50 cc. of a 1:10,000 solution given every three to four days produced satisfactory relief. This treatment was supplemented by the daily sublingual administration of 5 drops of 1:2,000 histamine solution. The patient has remained free of symptoms for about 15 months with no further treatment.

CASE 3.—H. K. H., a man, aged 43, reported for examination September 20, 1944, complaining of attacks of deep, boring pain, confined to the left frontal, ocular, and occipital regions and the region of the cheek. The attacks began in 1938, occurring one to two times weekly and lasting for two to three months. Subsequent attacks appeared only once or twice a year lasting two to four weeks. At the time of examination he had had attacks more or less constantly for a period of four months. Almost all the attacks occurred at night, coming on two to three hours after retiring. The attacks lasted one to two hours and there was some relief in the standing or sitting position. Sweating, tearing, watering and stuffiness of the nose occurred on the involved side.

The examination of the ears, nose and throat was negative. During an attack the sweating phenomenon was observed.

Injection therapy was begun with 0.10 cc. of 1:100,000 histamine solution every two or three days. This was supplemented by the sublingual administration of 2 drops of a 1:10,000 solution twice daily. A month later he reported that he was free of symptoms. He continued free of attacks for seven months, then they recurred. Upon resuming treatment the attacks again subsided for a period of

15 months. Then attacks returned again but were quickly controlled.

CASE 4.—Mrs. A. H., a woman, aged 34 years, was first seen on September 13, 1946, on account of attacks of pain in the right frontal, ocular and cheek regions of nine months' duration. At the onset the attacks lasted three or four weeks. Then she was free until five weeks before observation. The pain was very severe, occurred mostly at night after retiring and was accompanied by the typical sweating, tearing and nasal congestion phenomena. An attack was precipitated by drinking a glass of wine.

The examination of the ears, nose, sinuses and throat was negative. During an attack sweating and swelling of the temporal vessels was evident.

The injection of 0.20 cc. of a 1:1,000,000 solution of histamine relieved the attack at the time of the original examination. Subsequent injections of 0.30 to 0.40 cc. were given every two to three days, supplemented by the sublingual administration of 2 drops of the 1:10,000 solution twice daily. Individual attacks were controlled by two to three drops of the above solution. On one occasion an attack was markedly aggravated by 15 drops. For a period of two months symptoms were adequately controlled with the above therapy. On continued sublingual administration, she remained free of trouble for four months.

CASE 5.—J. J. S., a man, aged 57, came under observation on October 29, 1946, complaining of headache referred to the region of the left frontal sinus, eye, cheek, upper teeth, temple, mastoid and occiput of five years' duration. From the onset the attacks occurred at irregular intervals. One year ago an attack lasted three weeks. Then they occurred off and on until three weeks before examination. The pain was described as deep and boring. It came on regularly at night two to three hours after reclining. Recently he had three teeth removed with no relief of pain. The examination of the ears, nose and throat was negative. During an attack he had tearing of the left eye, congestion and watering of the nose. The temporal vessels were prominent and tender to pressure. The skin over this area was markedly red as compared to the uninvolved side and was covered with sweat.

An attack of pain was relieved by the subcutaneous injection of 0.10 cc. of 1:1,000,000 solution. Subsequent injections given every two to three days were gradually increased to 0.50 cc. of 1,1,000,000 solution. During this period he took sublingually 2 drops of a

1:10,000 solution twice daily. The night attacks disappeared as soon as treatment began. After two months he was entirely free of pain except for a mild occasional daily attack which could be relieved by the oral solution.

In the differential diagnosis, as Horton[4, 5] has emphasized, the syndrome of histaminic cephalgia should not be confused with classic migraine. Histaminic cephalgia is characterized by the occurrence of frequent short attacks of pain lasting from a few minutes to several hours. The attacks begin and end suddenly. They appear later in life than migraine and are not accompanied by nausea, vomiting and visual disturbances. In migraine there is usually a positive family history.

Histaminic cephalgia is readily distinguished from trigeminal neuralgia because of the absence of trigger zones. The attacks of pain are paroxysmal in trigeminal neuralgia and the pain areas are definitely localized at the end points of distribution of the first, second, or third divisions. Allergic headache is usually of the generalized type and is often associated with other manifestations of allergy.

Although pain referred to the region of the frontal sinus, the eye and the cheek is not uncommonly associated with nasal allergy with or without complicating polyposis, it should not be confused with histaminic cephalgia. In the 32 typical cases herein reported, allergy was not associated with the cephalgia in a single instance.

According to a recent presentation by Williams,[6] the vasodilating syndrome is included with vasomotor rhinitis, Ménière's disease and myalgia as part of his "syndrome of intrinsic allergy." Although the Sluder[7] syndrome covers a wider range of symptoms it has some of the characteristics of histaminic cephalgia. No doubt many of the patients observed by Sluder[7] were typical cases of histaminic cephalgia.

Finally, histaminic cephalgia should be differentiated from a variety of pathologic conditions in the eye, the nose and the paranasal sinuses which give rise to pain in this area.

In the treatment of the group of cases reported by Horton he employed a solution of histamine diphosphate containing 0.10 mg. of histamine base per cc. or a 1:10,000 dilution. Treatment was started with 0.10 cc., increased by 0.05 cc. at each injection until 0.50 cc. was given. In some cases the dosage was increased to 1.0 cc. The injections were given twice daily. With the occurrence

of flushing of the face with any given injection the dosage was reduced 50 per cent and then an attempt was made to increase it again. After the patient became free of symptoms an average of 0.50 to 1.0 cc. (0.10 mg.) of 1:10,000 solution was given as a maintenance dose 1 to 3 times weekly. Horton[2-5] pointed out that the average dose is from 0.35 to 0.50 cc. of the 1:10,000 solution. He has also had a great deal of experience in giving daily intravenous injections of a 1:250,000 solution of histamine. Injection is recommended at the rate of 20 to 60 drops per minute, requiring about 90 minutes for administration.

In the treatment of 112 patients with other types of headache Horton[4] reported no significant improvement.

In the management of our group of cases of histaminic cephalgia we adopted a somewhat different type of therapy because of the difficulty of getting patients to report twice daily for the injections. We found that smaller doses varying from 0.10 to 0.50 cc. of a 1:1,000,000 solution to 0.10 to 0.50 cc. of a 1:100,000 solution given every 2 to 3 days were effective in maintaining effective relief of symptoms. This was supplemented with sublingual administration using 1 to 2 drops of the 1:10,000 or the 1:1,000 solution once or twice daily. In some instances the patient found that individual attacks could be controlled by this alone. When the patient became entirely free of symptoms all treatment was discontinued. Some remained free while others had to take a maintenance dosage. As recently pointed out by Feinberg[8] there is a wide range of tolerance to histamine, therefore treatment is an individual problem. Practically all patients gave a history of having periods of complete freedom of symptoms before they had had any histamine therapy. In some cases, therefore, it is only necessary to treat the patient when a series of acute attacks begins.

Twenty-nine of the 32 patients received satisfactory results from treatment. Ten of the 32 patients has been subjected to surgical procedures with no relief. Eight had operations on the nose and sinuses. Two had an intracranial section of the trigeminal nerve.

Atypical Cases. Besides the 32 typical cases of histaminic cephalgia selected from 126 patients there was a remaining group of 94 which may be considered as atypical. On the whole the atypical patients had complaints which were very similar to those of the classical type. The headache was almost always unilateral and had the same distribution. The attacks were of varying duration, some being short and some very prolonged. A majority of the patients had

night attacks and in many instances the headache came on upon arising in the morning. Nausea and vomiting, visual disturbances and a positive family history were rare so that their classification as migraine did not seem justified. Alcohol frequently precipitated attacks. In some instances the headaches were more pronounced with the menses or pregnancy. On the whole the atypical presented a history of less severe attacks than the typical group. There was also no definite history of sweating, tearing, and nasal stuffiness. Because of the fact that a number of these patients responded to histamine therapy, the question arises as to whether it is justifiable to classify them as a mild type of histaminic cephalgia.

Although the area most commonly involved in typical cases is confined to the frontal, ocular and cheek regions, the pain is commonly referred to the temple, mastoid and occipital regions. Occasionally the vertex area is involved. This would suggest that any of the cranial vessels may be the site of reaction. One patient who had symptoms referred to the pharynx and larynx responded satisfactorily to histamine therapy. In another patient, the symptoms were referred to the parotid region with sweating over this area. Histamine therapy gave relief.

Nineteen patients of the atypical group did not respond to histamine treatment. Six were finally classified as migraine. Five patients had headaches and vertigo, which responded better to nicotinic acid. Seventeen atypical patients had good results from histamine treatment. Finally there was a group of 56 patients who did not remain under observation long enough to determine the effect of treatment. Some of these patients probably had relief from a few injections and did not return. Others became discouraged because of the failure to obtain relief, which was probably the result of improper dosage. In the group of 94 patients 19 had nose and sinus operations without relief of symptoms.

SUMMARY

In a group of 126 patients complaining of unilateral headache confined chiefly to the frontal, ocular and cheek regions, 32 were classified as typical of the so-called histaminic cephalgia syndrome described by Horton. This group of patients showed no pathologic condition in the nose, sinuses or eyes to account for the headache. These cases are readily distinguishable from migraine and trigeminal neuralgia. It is suggested that a milder type of histaminic cephalgia occurs in which the pain is not as severe and in which there is no demonstrable sweating or tearing, but perhaps some nasal congestion.

This relationship is suggested by the fact that many of these patients show satisfactory response to histamine therapy.

On the whole histaminic cephalgia, migraine and trigeminal neuralgia must be differentiated from a variety of pathologic conditions involving the nose, the paranasal sinuses, the eye and the surrounding regions which give rise to pain or headache in this area.

The establishment of Horton's syndrome of histaminic cephalgia should be accepted by the otolaryngologist as an important revelation, for it offers the solution to one of the most difficult problems with which he has to contend.

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XVI

POLIOMYELITIS AND THE OTOLARYNGOLOGIST

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Probably more research has been expended upon poliomyelitis than upon any other single disease entity, and with less results. We know that the disease is caused by a virus occurring in many strains; we know a good deal about its pathology and something about its pathologic physiology; we know some things about the treatment of its results; but how it is spread, how the virus usually enters the body, how to immunize against it, and how to combat the virus per se we do not know very much better than did Heine and Medin when they first described it.

Poliomyelitis is essentially a disease of warm weather. Originally it was more common in rural areas and in small towns; this characteristic still exists but its incidence in large cities has increased a great deal. It tends to occur in epidemics, which in some areas tend to recur more or less regularly with only occasional sporadic cases between epidemics. Contrary to the behavior of most infections, it does not afflict the areas of poverty and poor sanitation so much as the relatively well-favored spots, and possibly for the same reason it is rare among negroes and Mexicans.

The age incidence has changed gradually through the years. Early reports placed nearly all patients in the age group up to three or four years, but more recently older children and a constantly increasing number of adults have been afflicted. It has been suggested that this change in age incidence may be coupled with the apparent paradox of high incidence in areas of good sanitation and nutrition, on the assumption that good living conditions may protect the child during infancy when the disease tends to be abortive or mild, only to leave him unimmunized in later years.

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Its method of spread is not known. At present two theories on portals of entry are most favored, namely, the nasopharynx and the gastro-intestinal tract. The former is the older theory and is supported by analogy with many other infections, by repeated isolation of the virus from the nasopharynx, by the irritation often found here early in the disease, by the demonstration of lesions in the cranial nerves and their nuclei, and by the fact that it is possible to infect animals by this route. Gastro-intestinal entry offers many similar arguments in that it also is a common portal for infections, by the fact that the virus may be isolated from stools and from the intestinal mucosa, by the existence of gastro-intestinal symptoms early in the disease fully as often as throat symptoms, by the fact that incontrovertible milk-borne epidemics have been described, and further by the fact that at autopsy damage is found in all the abdominal and thoracic viscera, indicating a general involvement preceding and in addition to the characteristic changes in the nerve cells. A third possibility, less strongly supported but not excluded, is that an insect vector may be involved. This possibility hinges on the occurrence of the disease during the season in which insects are plentiful, plus the fact that the virus has been isolated from flies. None of these theories fits all the facts known about the disease and it may well be that there can be more than one portal of entry. At present, the gastro-intestinal route is favored by most observers.

The epidemiology of poliomyelitis is still an enigma. Person-to-person spread is tacitly assumed by the practice of quarantining patients and isolating contacts, but these measures have never availed in controlling an epidemic. Cases tend to be scattered over a wide area with no known contacts between the persons involved; multiple cases within a family are decidedly the exception and not the rule. When such cases do occur, the onset is usually at so nearly the same time that a common source of infection is suggested rather than a spread from one member to another. But neither does the pattern of an epidemic often suggest ingestion of the virus, as for instance occurs in typhoid fever; this despite the milk-borne epidemics mentioned and the fact that the virus has been repeatedly isolated from sewage. If it is insect borne, it is hard to see how those parts of the community with the fewest insects should so commonly have the most poliomyelitis. In any event, nothing that has ever been done by any health department has ever altered the course of an epidemic in the slightest degree, and some states have already removed all restrictions on these patients.

Since public health measures of control are ineffective, attempts have been made to immunize individuals. One of the first of these was an attempt to render the olfactory area impervious to the virus by treating it with cauterizing agents. Protection afforded by this measure is hard to evaluate, since the incidence of the disease is so low that enormous series of patients would be needed for convincing evidence. Since the procedure caused considerable local irritation and sometimes anosmia and especially since there came to be increasing doubt as to the nasal portal of entry, it was abandoned.

Then there were efforts to produce active immunity by the injection of attenuated virus and of killed virus. The first did produce demonstrable immunity, but also it unfortunately produced the clinical disease in a proportion at least as high as the normal expectancy in time of an epidemic. The second, as might be anticipated, was inactive.

Passive immunization through the use of convalescent or pooled adult serum has a certain logical appeal, but no mass tests have ever been possible to prove its worth. Animals can be protected in this manner, but only if the serum is given practically at the moment of infection. It seems impossible that it could be available for a community test.

Certain individual measures of protection are available in that predisposing causes seem to exist in the form of unusual fatigue or exposure. For a time, the beach and swimming pools were strongly suspected as areas of dissemination, but it now appears that it is the strenuous activity and chilling incident to day-long use of these places that is to blame for their high incidence of cases. Disease frequently follows violent exertion in less crowded places and in the absence of water. Even the part of the body most used may be most seriously involved, as the right arm of a tennis player.

Beyond question many of the mysteries just indicated could be solved if the clinical cases were easier to recognize. The full-blown case is usually easy to diagnose, but for every such fairly obvious one there are a great many abortive cases incapable of diagnosis by any practical means now known. In addition, there is a host of healthy carriers, so that it is probable that during an epidemic a community is well saturated with the virus with only an occasional case with demonstrable nerve involvement being recognized. Thus we are in somewhat the same position we might occupy in regard to measles if the only case we could recognize were the one with complicating encephalitis.

The disease process tends to begin after about ten days' incubation. The first symptoms may be those of an upper respiratory infection, a gastro-intestinal upset, the ubiquitous "flu", or some combination of these. The patient is usually not very sick. His temperature is elevated but ordinarily it is not very high, and neither physical examination nor laboratory tests offer anything diagnostic. These vague symptoms may persist for a few days and disappear with complete recovery of the patient, or they may merge into the recognizable phenomena of nerve involvement, or they may disappear only to come again after about a week to form the biphasic or dromedary type of the disease.

Neural symptoms usually begin with stiffness of the neck, back, or posterior leg muscles, singly or more often in combination, and with more or less lack of symmetry. These muscles are sore and tender to deep palpation, and also to movement and particularly to stretching. This tightness and soreness of muscles is not due to mere meningeal irritation, for they appear in erratic asymmetric patterns incompatible with that cause.

Concurrent with these changes or shortly after they appear, weakness begins and may exist in all degrees from the barely detectible to utter paralysis. This weakness is most common and most severe in the extremities and in the opponents of tight muscles, and is often greatest in muscles recently subjected to severe exertion; however, it may involve any of the voluntary muscles. Other changes of diagnostic importance are found in the reflexes. Usually the superficial reflexes are affected first and then the tendon jerks are diminished or abolished.

Spinal fluid changes usually afford diagnostic confirmation after nerve changes have begun, but it is important to note that unmistakable poliomyelitis may occur with the spinal fluid normal on repeated examination. Such findings have been reported in as many as 30 per cent of cases in some epidemics. The expected findings are moderately increased protein and pleocytosis, chiefly lymphocytic, of 50 to 200 or 300 cells with occasional counts of several times this number.

About a week after neural changes have begun, the fever and the constitutional symptoms disappear and no further damage occurs save in the occasional patient who relapses. From this point on some improvement may be expected in every patient, whether treated or not, or whether treated by one method or another. The degree of recovery which occurs spontaneously or is obtained by treatment

is of course determined by the extent and the severity of the nerve damage inflicted, and this may be anything from a quickly reversible edema of the nerve cells to complete necrosis.

The first and only attempt at specific treatment was the use of convalescent serum. Its results were not statistically impressive and it has been almost universally abandoned. In defense of this type of treatment it may still be said that different sera are known to possess widely varying amounts of antibody and that to date there has never been an adequate trial with serum of proven high potency.

Sister Kenny has caused great public furor as well as sharp medical controversy, and it would be venturesome to attempt an exact appraisal of the value of her teachings. There are, however, some observations which most students of the disease would accept. In the first place, she undoubtedly gave a tremendous impetus to research in therapy and suggested new or at least previously ignored lines of approach. For instance, she insistently drew attention to the functional importance of tight muscles, which had always been known to exist but which had been considered of relatively little importance. She also offered a treatment for these tight muscles which, laborious and illogical as it may seem, must be admitted by any unprejudiced observer to have definite therapeutic value. By this same insistence on the importance of tight muscles she made us look for them, which resulted in the diagnosis of milder and earlier cases. Her methods did much for the comfort of patients by hastening the abandonment of splints and casts, which in retrospect had little to commend them. Her end results have apparently been better than the usual results of previous methods.

The first effect of her teachings was to stimulate a search for a better and simpler method of relieving muscle spasm. Prostigmine gave some promise of doing this, but its results were erratic and unpredictable. More recently, curare has been tried and present evidence suggests that it may be a decided advance. Used alone, it results only in a period of comfort to the patient, in that it seems to cause the affected muscles merely to cease to contract rather than actually to relax. However, curare plus rather vigorous physical therapy during its period of action may accomplish a definite and rapid return of the muscles to normal length. It also is occasionally of dramatic help in those respirator patients who retain some ability to breathe for themselves but who cannot synchronize their efforts with the rhythm of the respirator. In such patients the

remaining voluntary respiration is temporarily inhibited by the cuare until the patient learns to breathe with the machine instead of fighting it.

Another innovation in treatment is tracheotomy in certain cases with respiratory involvement. Since death in poliomyelitis occurs only from anoxia or conditions associated with it, it is obvious that these patients represent the most acute problems in the whole field of therapy.

Patients with bulbar lesions may have a variety of respiratory problems. Quite frequently the difficulty may be only in the throat, with inability to swallow but with little or no true respiratory paralysis. These patients may be mistakenly put into respirators to their very great detriment. If they are given postural drainage plus suction as needed, they do very well, since paralysis of these muscles shows a high percentage of recovery. Intrinsic laryngeal involvement also occurs, both with spasticity and flaccidity of muscles.

True respiratory involvement may include the chest muscles, the diaphragm, or both. Diaphragmatic paralysis is the more ominous. Candidates for both the respirator and tracheotomy come from this group.

Indications for tracheotomy are not clear-cut, and possibly they may never be. Physical signs of anoxia are scant in the early stages of the disease, and even when they are marked or can be accurately followed with the oxyhemoglobinometer it may still not be easy to determine whether these patients would be relieved by operation. It is certain, however, that death is often not due to failure of the respiratory muscles alone, but to this plus secondary changes in the lungs and the bronchial tree such as mucus plugs, atelectasis, pulmonary edema, and hemorrhage. Many of these can be approached via the tracheotomy tube and bronchoscopy, and at least an occasional patient may be given the added time needed to make a good recovery from his primary illness. Our own clinic plans to report its recent experiences in the near future.

Undoubtedly the most vexing question regarding poliomyelitis for the otolaryngologist is the matter of the "closed season" on tonsillectomies at the time of an epidemic. Valid mass statistics on this question are almost impossible to obtain. It is hard to determine the number of tonsillectomies done in a large city in a given period of time, much less the number of cases of poliomyelitis; hence the relation of the operation to the incidence of the disease has been studied

on the basis of admittedly incomplete data. At present one may question whether a tonsillectomy makes a subsequent attack of poliomyelitis any more probable. There are, however, impressive instances on record indicating that when such a subsequent attack does occur it is much more likely to be of the bulbar type. Even if one refuses to admit the scientific worth of these instances, a thing hard to do under the circumstances, there still remain other factors not to be ignored. When one considers that tonsillectomy is seldom a very urgent operation and when one also considers the public attitude toward this disease, it would seem emphatically the part of wisdom to postpone such an operation.

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XVII

PREMEDICATION IN TONSILLECTOMIES IN CHILDREN UNDER SIX YEARS OLD

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A review of cases of 150 children under six years of age has been made to try and evaluate the advantages and disadvantages obtained in administering preanesthetic sedation such as the barbiturates, and in substituting scopolamine for atropine previous to tonsil operations.

There have been about 200 children under six years of age operated on with this premedication over a six-year period, from 1940 through 1946, at the New England Hospital for Women and Children, Roxbury, Mass.

The importance of maintaining a completely relaxed throat is discussed and the question raised as to the possibility of maintaining the necessary depth of anesthesia with ether when the barbiturate and scopolamine are used in combination, without causing anoxia. My experience has been that it is difficult to accomplish.

Hawksley[1] feels sufficient depth of anesthesia must be reached and maintained for the surgeon to remove the tonsils unhurried from a relaxed throat, and that it is also important to maintain this relaxation while the adenoid is being removed.

Fish[2] expresses the general feeling about tonsillectomies in children by saying it is considered the least hazardous of operations on children. Yet from the viewpoint of anesthesia there is no operation which requires more skill and judgment.

An editorial by Dittrick,[7] in the same publication says, anesthesia can be deadly. A number of unfortunate tendencies have developed in the field of anesthesia. These tendencies increase as the gap in knowledge between the trained anesthetist and the occasional anesthetist occurs.

Galloway[3] discusses anoxia, stating, "The trained anesthesiologist is alive to the hazards of anoxia. However, a feeling of undue confidence may develop. Serious consideration should be given the heavy

levels of premedication advised before general anesthesia. Cyanosis may be a late sign of oxygen need."

Courville[4] believes changes in the brain may occur after three to eight minutes of anoxia. In calling attention to the serious results which follow anoxia of the brain from nitrous oxide anesthesia, he says, "Several factors may be present, all contributing to the production of a cerebral lesion. The effects are due to asphyxia and not to any toxic effect of the nitrous oxide itself." He mentions the exposure of the brain to a dangerous degree of oxygen want and calls attention to occasional delayed symptoms due to asphyxia of the cortical nerve cells.

The word anoxia has come to signify any decrease in the normal percentage of oxygen in the blood. It is surprising to find so little attention paid to anoxia.

Evans,[5] writing about the effects of anoxemia following central nervous depression, states that obstruction of the upper respiratory passages following the depressing effects of preanesthetic drugs or of the anesthetic itself may be the cause of postanesthetic anoxemias.

The causes of anesthetic anoxemia are relaxed tongue, relaxed epiglottis, mucus or blood obstructing the cords and contributing to laryngospasm. The chief necessity in the treatment of anoxia is to supply oxygen to the blood.

Cullen[6] mentions among other functions of premedication the counteracting of the undesirable side-action of an anesthetic agent. He feels that the dosage of scopolamine required in those under five and those over seventy-five years of age may induce unfavorable reactions. Cullen also states that the barbiturates sometimes cause respiratory depression, obstruction and asphyxia, causing shallow respiration. Another unfavorable reaction of the barbiturates is that they increase the sensitivity of the laryngeal reflex and predispose to laryngospasm; for this reason they should not be used as premedication for ether. If the anesthetist is unaware of the possibilities of laryngospasm, spastic adduction of the vocal cords can be dangerous. Once present it must be treated promptly, the object being to supply oxygen.

Routine. All patients entered the New England Hospital the afternoon before the operation. Throat cultures, temperature and histories were taken and physical examinations were done. The laboratory did hemoglobin, clotting time, bleeding time and urine tests on all patients. Each patient was visited in the evening by a

member of the anesthesia department and premedication was ordered after an evaluation of the patient's condition. The dosage to be given was ascertained by the weight, height, age and general condition of the patient.

In 1939 only seconal or nembutal, in minimum doses, was used as a sedative. Atropine sulphate was given. This one narcotic drug, given in about one-half the dosage shown in the table, proved more desirable to the surgeon as it seemed to result in fewer reactions. The barbiturate was administered by rectum, in capsule form. Cullen[6] believes the barbiturates to be respiratory depressants.

In 1940 the premedication was increased to the following dosages:

WEIGHT CHART AND DOSAGE

PREOPERATIVE

AGE	SODIUM SECONAL	SCOPOLAMINE	WEIGHT
2 yr.	gr. 1½	gr. 1/300	25-30 lbs.
3 yr.	gr. 1½	gr. 1/300	30-35 lbs.
4 yr.	gr. 3	gr. 1/250	35-40 lbs.
5 yr.	gr. 3	gr. 1/250	40-45 lbs.
6 yr.	gr. 3	gr. 1/250	45-55 lbs.

In 1944 the anesthesia department took full control and the responsibility of ordering all the premedication. No morphine sulphate was given in this group. Seconal was the barbiturate used and occasionally devinal was substituted or added in fractional dosage. It was a requirement of the anesthesia department, if house anesthesia was used, that its members order the premedication. Cooperation is necessary in a general hospital, when there is a well-organized registered anesthesia department.

Galloway[3] says the laryngologist should not delegate the entire responsibility and must be alive to the hazard of anoxia in anesthesia.

All ether charts of the cases reviewed were found to be fully completed. These were private patients. The drug dosage and the time the anesthesia was started, the time of beginning the operation and the finish were recorded. The average time of induction was 20 to 25 minutes and the average time of operation was 20 minutes.

A sample case is that of a child, four years old, weight 40 pounds, who would be given seconal, gr. 3, in capsule by rectum, and scopola-

mine, gr. 1/250, hypodermically one and one-half hours preoperatively.

One of the disadvantages of the administration of the basal premedication for tonsil operations is that it increases the hazards. There is the necessity for a well-trained staff of anesthetists. It is necessary for the surgeon to become familiar with the increased hazards and to learn to observe and to evaluate quickly any unfavorable change in the patient's condition. There is usually an increased time for induction. A disadvantage, also, is the administration of unnecessary drugs to children. The uncertainty of the reaction is another disadvantage. Two children of nearly the same age and weight may react quite differently to the same appropriate dosage. One will come to the operating room awake and talking, the other will be sound asleep. One will develop laryngospasm[6] at the beginning of the anesthesia and another at the start of the operation or when some blood comes in contact with the larynx. Some will breathe so quietly it is necessary to listen carefully to hear the respiration. This shallowness of respiration and laryngospasm are what cause the anoxia.

In cases of postoperative bleeding there is danger of the child remaining too quiet, from drowsiness, during the afternoon following the operation. There were two postoperative pharyngeal hemorrhages among these cases. One child was four years old and had been asleep until visited by her general physician about 4:00 P. M. (six hours after the operation). Her condition at that time was thought to be good. The pulse was rapid, there had been no fresh bleeding or vomiting, yet a short time after this she vomited a basin of dark blood and soon vomited another. On examination by me a large clot was found filling the pharynx and extending down so far that the blood was dripping from it and running down the throat without the necessity of swallowing. The clot was removed and postnasal packing placed. The child had lost so much blood that a transfusion was considered advisable.

There is the possibility of inhalation of blood into the trachea with chest complications resulting, if a patient is allowed to sleep quietly after the operation with the cough reflex abolished. A patient must be watched and turned frequently.

The necessary preparation and the care which is required in ordering premedication, especially in children under five, seem a disadvantage especially if there are five or six operations to be done

in quick succession. Each operation, which in itself takes only 20 minutes or less, acquires the proportions of a major procedure.

The advantages, from the standpoint of the anesthetist, are a quiet, nonapprehensive patient for induction. The advantage from the psychiatrist's and the surgeon's viewpoint is postoperative amnesia when scopolamine is used. Children remain amnesic from the scopolamine for several hours. This was illustrated in a child of five whose aunt was a nurse in the Hospital. She went to see him several times in the afternoon. Once, about five hours postoperatively, he wakened and spoke to her. As he let out a couple of lusty yells she told him his throat would be sore if he did that. He replied that it was sore now. In the morning this very bright and well-behaved child insisted he had not seen his aunt. The mother also is pleased that the child has no remembrance of the application of a general anesthetic.

The older patient remembers being moved from his bed to the operating room. He usually remembers speaking to the surgeon before operation. It is doubtful if the child under five remembers even this. After that the adult patient remains unaware of anything that happens for the next five or six hours.

DISCUSSION

There is a wide gap in the experience of the trained anesthetist and the surgeon. It is only by long experience that a surgeon has confidence in a new method of anesthesia, especially when the method seems to raise some hazards. When the surgeon thinks a patient is in danger, the surgeon is not operating under the best circumstances. There may be a sensation of the need for haste to bring to an end a difficult situation.

Anesthesia for throat operations must be of considerable depth in order to overcome the throat reflexes. This relaxation is necessary for in applying the snare to the tonsil it is easy to catch the pillars and partially amputate them if the throat is not relaxed. Bleeding occurs more frequently if the posterior superior angle of the posterior pillar is caught and the pharyngopalatine muscle is injured. Also, if the pharynx is active while the adenoids are being removed, injury may be done to the superior constrictor muscle of the pharynx or to the eustachian tubes. The basal anesthetics do not relax the throat reflexes, which makes it necessary to administer as much general anesthesia, in the form of gas oxygen and ether, as if there had been no basal anesthetic administered. During induction

of a general anesthetic, after premedication, the child breathes gently, with rather shallow respirations, and slips easily into unconsciousness in a few minutes.

Preparation for the surgeon to start the operation is completed in from 15 to 25 minutes. In the majority of cases, as soon as the tongue is depressed, there is a gag reflex and it is necessary to continue the inhalation anesthesia another five minutes to complete the pharyngeal relaxation. As the anesthesia deepens a certain percentage of patients become cyanotic, with some anoxia. The pulse usually remains good. The respiration is shallow. It becomes necessary to add oxygen to the ether to counteract the depressing action of the basic anesthetics. In a certain percentage of patients a laryngeal spasm develops. The administration of oxygen will usually correct this. Care is necessary to prevent blood from entering the larynx. If the patient is on the edge of anoxia and the surgeon puts heavy pressure on the tongue and so reduces the amount of oxygen or allows blood to run down and irritate the larynx, it may bring on laryngospasm and anoxia. There is a disadvantage to the surgeon if sufficient depth of relaxation cannot be maintained because of anoxia and inability of the patient to tolerate continuous administration of the ether. The generalized lack of muscular tone in the under-five-year-old group was noticeable. The average patient, on returning to bed, sleeps until late in the afternoon. If there should be postoperative bleeding the nurse is likely not to detect it until a stomachful of blood is vomited up. Then the pulse is found rapid. The child becomes restless and bright blood is expectorated or there is another vomiting. A third vomiting is to be reported to the surgeon in all cases.

CONCLUSIONS

If basal anesthetics are used preoperatively in tonsillectomies, great skill and care are required to judge the dosage.

It is necessary to have well-trained anesthetists with experience in throat operations.

The dangers from laryngospasm and anoxia seem very real.

To obtain relaxation of the throat, as deep an anesthesia is required from the general inhalation anesthesia as if no basal premedication had been given.

The disadvantages far outweigh the advantages.

After five years of experience with this required premedication, I recommend eliminating the barbiturates given in combination with scopolamine as too depressing to respiration. Atropine can be used and a basal anesthetic in minimal dosage.

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XVIII

DEAFNESS DUE TO ACOUSTIC TRAUMA IN WARFARE

ANALYSIS OF 72 CASES

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Impairment of hearing due to the effect of exposure to loud or prolonged noises has been a recognized clinical phenomenon for many decades. In World War I considerable data were collected on cases due to explosives and large caliber gunfire.[1] In World War II a higher incidence of traumatic hearing impairment occurred despite cognizance of the cause and attempts at protection. This was to be expected in view of the great increase in fire power of the large number of rapid-fire small and medium caliber guns, the violent detonation of heavy caliber cannon, the intensified blast of large shells and huge bombs carrying the more powerful new explosives, and the frequency of traumatizing noises encountered in the modern mechanized armamentarium, including tanks, diesel engines, and high-powered aircraft.

In industry the harmful effects of excessive noise on auditory acuity has long been known. War is a gigantic industry in which the multiplication of the number and intensity of detonations and of loud continued noises has resulted in an increasing number of aural casualties with impairment of hearing. Various phases of this subject have been elucidated in recent articles.[2-9]

This paper will present 72 cases of deafness due to acoustic trauma, with an analysis of pertinent data by means of tables and representative audiometric graphs. Prevention and treatment of the condition will be discussed briefly.

Table 1 lists the causes of aural trauma in this series.

The injury resulting from the traumatic effect of noise has been shown to be a degeneration of various areas of the organ of Corti. Moderate damage is limited to the basal coil, corresponding to the

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TABLE 1.—CAUSES OF ACOUSTIC TRAUMA.

	CASES
Large caliber gunfire (3 in. to 155 mm.)	33
Aerial bomb	15
Medium caliber gunfire (20 mm. to 40 mm.)	5
Rifle fire .30 caliber	5
Explosion (ammunition dump, ship, etc.)	5
Pistol fire .45 caliber Colt	4
Mine (floating, land)	2
Large caliber shell burst	1
Bangalore (land) torpedo	1
"K" type depth charge gun	1

tones above 2048 d.v. Severe trauma affects the entire tone range in varying degrees up to total deafness. Some changes are transient; others are irreversible or even progressive.

Pathologic changes in the human labyrinth due to the effect of sound have been described by Wolff.[10] The basal coil of the cochlea was presumed to be the most vulnerable by virtue of its exposed location in relation to the round and oval windows and the peculiarity of the vascular system in this area.

Lurie and his co-workers[11] made comprehensive microscopic studies of the cochleas of guinea pigs exposed to traumatizing intensities of sound. In ascending order of severity the following lesions were found: Loss of mesothelial cells, change in the cytoplasm of the hair cells, degeneration of the hair cells, rupture of the organ of Corti, dislocation of the organ of Corti from the basilar membrane, and complete disappearance of the organ with degeneration of the cochlear nerve fibers and of the cells of the spinal ganglion corresponding to the damaged area. When caused by low tones, the damage was noted nearer the helicotrema; when caused by high tones, it was near the round window.

Table 2 gives a statistical review of the cases examined. Study of the audiograms reveals no characteristic graph. Fig. 1 illustrates representative curves. The most constant curve was seen following moderate trauma. This presented a V dip of variable depth. Of the 123 graphs indicating hearing loss, the decline centered at 5792 d.v. in 9 curves (7.3 per cent), at 2896 d.v. in 14 curves (11.3 per cent), and at 4096 d.v. in 46 curves (37.4 per cent). In 15 graphs (12.2 per cent) the loss was equal at 2896 d.v. and

TABLE 2.—STATISTICAL ANALYSIS OF 72 CASES OF DETONATION DEAFNESS

		NO. OF CASES	% OF CASES
Deafness	Unilateral	18	25
	Bilateral	54	75
Degree of Deafness	Slight	13	18.1
	Moderate	33	45.8
	Severe	26	36.1
Tinnitus	Transient	14	19.5
	Persistent	56	77.7
	Total	70	97.2
Perforation of tympanic membrane	Present	8	11.1
	Healed	15	20.8
	Total	23	31.9
Otitis media	Transient	6	8.3
	Chronic	2	2.8
	Total	8	11.1
Pain at time of trauma		27	37.5
Bleeding from external auditory canal		13	18.5

4096 d.v. The latter frequency appears to be the most vulnerable to acoustic trauma, with a combined incidence of 49.6 per cent.

Also, 73 curves (58 per cent) showed a notable secondary decline at 11584 d.v., after recovery from the dip at 5792 d.v. and 8192 d.v. This may be considered to be an intermediate stage between a V notch and a severe loss in all frequencies above 2048 d.v.

The remaining audiograms portrayed a varied pattern of deafness in proportion to the degree of trauma. When more severe, the V notch broadened to include adjacent frequencies between 1024 d.v. and 8192 d.v. The most severe cases showed greater losses up to total deafness. Moderate decreases in the frequencies above 2048 d.v. usually caused little noticeable handicap for useful hearing.

Tinnitus was observed to be the most constant symptom in acoustic trauma, occurring in 70 cases (97.2 per cent) in my series. Henry[12] reported an incidence of tinnitus in 42.4 per cent of cases and Boyes-Kirkis[13] in 90 per cent of cases. However, the relationship between the degree of deafness and the intensity and persistence of tinnitus was inconstant.

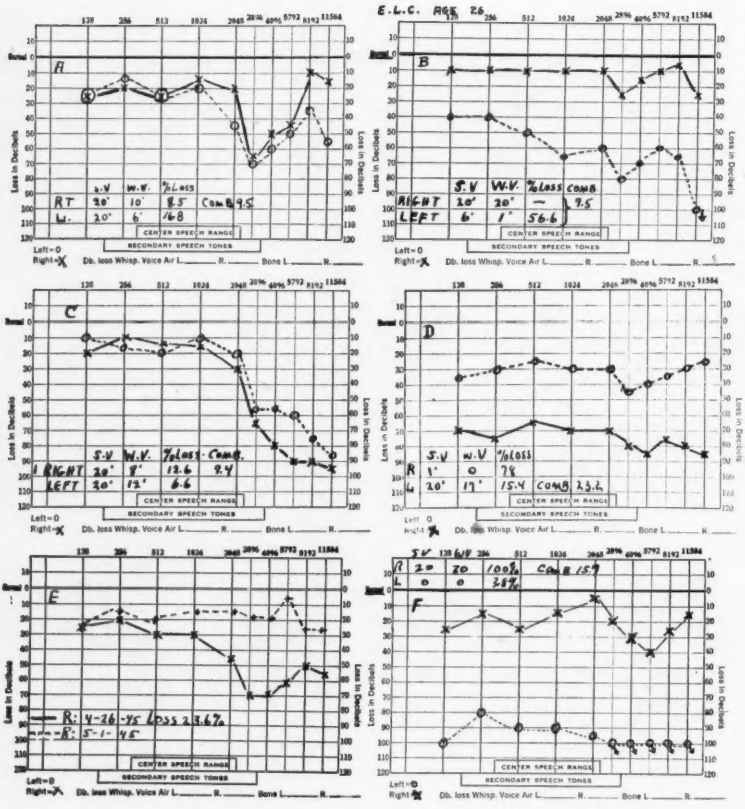
A
S.V W.V. %Loss
RT 20' 10' 85 Comb 9.5
Lt. 20' 6' 68
B
E.L.C. Age 26
S.V W.V. %Loss comb
RIGHT 20' 20' — 9.5
LEFT 6' 1' 56.6
C
S.V W.V. %Loss Comb.
RIGHT 20' 8' 12.6 9.4
LEFT 20' 12' 66
D
S.V W.V %Loss
R 1' 0 78
L 20' 17' 15.4 Comb 23.2
E
R: 4-26-45 Loss 23.6%
L: 5-1-45
F
S.V 128 6/8 256 512 1024 2048 2896 4096 5792 8192 11584
R 20 20 100% Comb 15.9
L 0 0 28%
Loss in Decibels
CENTER SPEECH RANGE
SECONDARY SPEECH TONES
Left=0
Right=X
Db. loss Whisp. Voice Air L. R. Bone L. R.

Fig. 1.—Audiograms of cases of auditory decrement due to acoustic trauma. Crosses indicate the right ear; circles the left. Percentage losses were calculated by the method adopted by the Council of Physical Therapy.[27]

A. Age 32. Member of 40 mm. gun crew, firing almost daily for three months. The drums are normal. Interval of five months between trauma and examination. Tinnitus is slight.

B. Age. 26. Served a 5 inch gun on a battleship during Pearl Harbor attack, 22 months preceding examination. Rupture of the left tympanic membrane with subsequent chronic otitis media and persistent tinnitus.

C. Age 22. Rifle range instructor for six hours daily, five days a week for 13 months preceding audiometer test. No ear protection. Drums are normal. Tinnitus present for first three months only.

D. Age 38. Patient was knocked down by blast of a 155 mm. gun, 16 months preceding examination. Tinnitus, headaches and vertigo persist. A dry perforation is present in Shrapnell's area of the right drum. The left drum is normal.

E. Age 30. Hearing loss following Colt .45 caliber pistol practice for one day. Spontaneous recovery in four days. Drum is normal. Moderate tinnitus.

F. Age 21. Patient was knocked down by the blast of a mine while on deck, 6 feet above waterline. The drums are intact. Tinnitus is moderate. Interval between trauma and audiometer test was two months.

The occurrence of rupture of the tympanic membrane in 31.9 per cent of the cases in my series is low in comparison to the observations of others, which have been reported as follows: Silcox and Schenck,[14] 70.7 per cent in 82 cases; Boemer,[15] 59.1 per cent in 115 cases; Henry,[12] 52.4 per cent in 292 cases. These higher percentages may be attributed to examination after a shorter interval following injury, when recently healed perforations could be recognized more readily.

Otitis media occurred in only 8 cases in my series (11.1 per cent). There was no complicating mastoiditis. Boemer,[15] however, reported an incidence of 21.7 per cent.

No parallelism was noted between the deafness and the concomitant symptoms and findings. In a number of cases severe deafness was associated with a normal tympanic membrane (Fig. 1F). Conversely, rupture of the drum occurred in two cases with only slight loss of hearing. One individual 18 years of age had been exposed to the firing of a 3 inch gun. When examined seven days later, he complained of slight tinnitus. The right drum was normal. The left drum exhibited fine capillary injection with a recent perforation in the anterior-inferior quadrant covered by a thin membrane. The audiogram revealed very slight decreases, averaging 14 db. on the right and 15 db. on the left with a maximum loss of 20 db. at 4096 d.v.

Perlman[16] listed the following factors which may influence the degree of auditory loss: (1) total time of exposure; (2) length of each exposure; (3) loudness of sound stimulus; (4) age of subject; (5) constitutional factors; (6) type of sound, constant or sharp; (7) use of protective devices; (8) exposure in closed or open spaces; (9) previous aural disease; (10) frequency of sound stimulus. Unfortunately, it is not feasible to determine all of these factors in most cases.

Figure 2 illustrates a scale of intensity of various sounds.[17] Proximity of the subject to the noise determines the degree of trauma. Critical distances vary with the type of noise.

Referring to sustained noises, Bunch[18] declared: "The boundary line between noises which cause hearing losses and those which do not lies between 80 and 90 decibels." However, a single detonation must be higher to produce trauma. According to Behnke,[19] permanent injury to the inner ear may be caused by a noise intensity of 120 decibels. Sutherland, quoted by Behnke, estimated the noise level

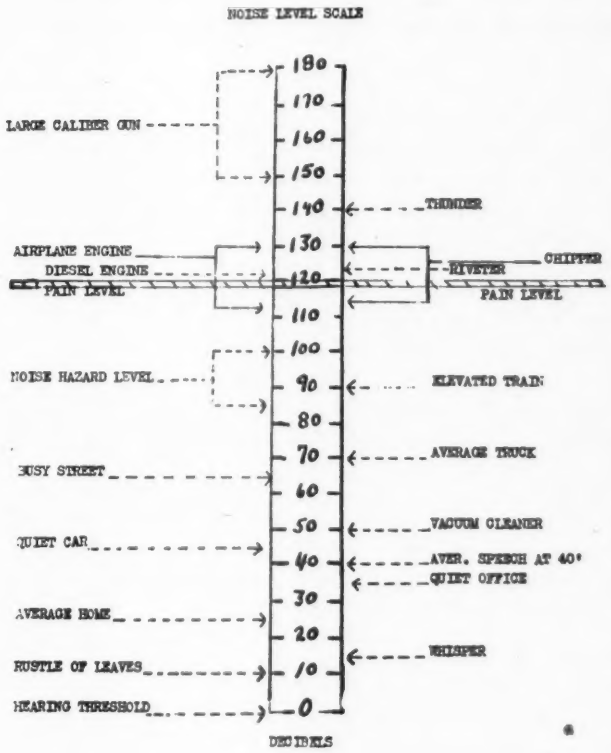

Fig. 2.—Scale of noise intensities. Modified from McCoy.[17]

of the firing of a large caliber gun to be from 150 to 180 decibels' intensity at the moment of detonation.

Variations in the susceptibility to blast have been noted by many observers. Lurie[20] has stated, "Changes in the organ of Corti are reversible if they do not go beyond a certain point. Beyond this point the organ degenerates and normal function is lost."

In a series of 121 cases, Truex[21] observed three cases of recovery from severe auditory decrement resulting from blast. However, he commented, "There is no accepted explanation, either physiological or pathological, for the injury and subsequent recovery process in some cases of traumatic nerve deafness."

Cantor[22] has advanced an ingenious hypothesis explaining the mechanism of blast effect on the tympanic cavity, drum membrane and inner ear. On the basis of the physical principles involved he concluded that the pathway of blast is through the eustachian tube, with exertion of pressure on the medial side of the drum with resultant outward rupture. He concurred in the opinion that rupture of the drum has a protective influence in diverting the blast outward, thereby diminishing the amount of cochlear damage. His theory gains support from the evidence that fresh traumatic perforations show everted edges and are usually in the anterior inferior quadrant of the drum, opposite the tubal orifice.

He also quotes observations that in cases of unilateral perforations the greatest hearing loss is in the ear with the intact drum. This is at variance with my findings. In 16 of 20 cases of unilateral rupture of the drum, the greater hearing loss was noted on the same side as the perforation.

Furthermore, there was no significant relationship between the degree of auditory loss and the state of the tympanic membrane. The findings are variable. However, these inconsistencies do not entirely discredit Cantor's plausible concept.

There has been some uncertainty as to whether the detonation (sound) or the blast (pressure) causes the damage to the auditory apparatus. Thompson[23] has clarified this point by indicating that sound waves of very great condensation may become shock waves as a result of propagation phenomena. However, this explanation apparently does not apply to hearing decrement following continued exposure to noise, as in industry or in experimental use of a constant tone, with resulting fatigue or eventual degeneration of a specific zone of the cochlea.

Boyes-Kirkis emphasized the distinction which must be drawn between impairment of hearing due to constant exposure to the noises of battle and that due to sudden intense acoustic trauma from blast.

Management of cases of deafness due to acoustic trauma may be divided into three phases: treatment, rehabilitation and prevention.

Treatment of the aural damage is limited in scope and is largely ineffective in so far as restoration of the hearing is concerned.

The ruptured tympanic membrane should be guarded by a plug of sterile cotton. Blood clots may be removed with a forceps. There is occasional indication for judicious installation in the external canal of either microcrystalline sulfathiazole or a solution of penicillin containing 1000 units per cc. Frequent examinations or any irrigations of the auditory canal are to be avoided.

A complicating otitis media requires the usual local management plus systemic penicillin therapy or chemotherapy.

Tinnitus may be benefited by sedatives, by inflation to insure the patency of the eustachian tubes and by concomitant psychotherapy. Daily oral administration of 5 mg. of thiamine hydrochloride and 25 mg. of nicotinic acid for an extended period may have some value.

A psychiatric interview may be indicated to evaluate functional factors due to combat fatigue or hysteria, or to discover signs of associated psychic trauma.

It is also necessary to be alert in exposing the occasional case of volitional deafness by the various tests for malingering. Hipskind[24] employed the aid of other than medical personnel in the observation of suspects.

The degree of spontaneous recovery is unpredictable. In cases with slight impairment complete recovery occasionally occurs (Fig. 1E). In moderate hearing losses some gradual improvement may be noted, but this is not to be expected if it is not apparent in the first two to eight weeks. In severe cases the initial loss is usually permanent and constant. In others, a progressive loss of hearing may continue for a period of months.

Discussion of rehabilitation and reeducation of the partially deaf individual is beyond the scope of this paper. A comprehensive program was established by Lederer at the Philadelphia Naval Hospital for study of these cases, with attention to lip-reading instruction, fitting of hearing aids, vocational training, educational guidance and psychologic appraisal. He and his associates[25] have reported at length on all phases of the subject. The Army has three general hospitals where the deaf receive special care.

Prevention of acoustic trauma is of major importance to reduce the growing number of aural casualties. This involves education of all military personnel as to the occurrence, emphasizing the neces-

sity for protection and giving instruction in the methods of guarding the ears. Use of cotton in the ears is only partly protective, perhaps because of faulty insertion in some cases. Specially designed ear plugs afford adequate safeguard. Under the auspices of the National Defense Research Council, the Psycho-Acoustic Laboratory of Harvard University devised an ear plug called the "Ear Warden" (Type V-51R), which is moulded of soft black neoprene, a synthetic rubber. This material, or a soft vinyl plastic, was found to be practical from the viewpoint of ease of insertion, cleanliness, durability, low cost, feasibility of mass producton and comfort to the wearer. Extensive testing demonstrated the efficiency of the plug.[26]

The modus operandi of the plug is the reduction of the intensity of the sound wave below the level injurious to the auditory mechanism.

General use of ear plugs would have been instrumental in reducing the large number of cases of hearing impairment. It is regrettable that this was not uniformly practical because of the exigencies of warfare, the large number of personnel involved, and the not infrequent refusal to wear the plug.

SUMMARY

1. Seventy-two cases of deafness due to acoustic trauma from detonation have been studied, with an analysis of hearing data, clinical signs and symptoms by means of tables and audiograms.

2. Treatment has been discussed with emphasis on prevention.

3. The valuable information acquired in warfare should be applied extensively in future military training programs and in peacetime industries where detonations or continuous noises afford a hazard to the hearing function.

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Clinical Notes

XIX

LARYNGEAL POLYPOID GRANULOMA FOLLOWING INTRATRACHEAL ANESTHESIA

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The increasing use of the endotracheal tube in anesthesia has been reported to be without serious sequelae. The following case is reported to again call the attention of the medical profession to the possibility of trauma to the larynx during intratracheal anesthesia and its possible serious complications.

Mrs. J. S., aged 49, was admitted to the Montefiore Hospital on March 6, 1946, with complaints of fatigue, vomiting and tarry stools. The patient had had a thyroidectomy two years previously and became increasingly tired and fatigued following the operation. Vitamins, iron and liver injections were tried to no avail. While taking this medication she had black stools but ascribed this to the iron therapy. The patient had begun to vomit about two months prior to admission, and the vomitus consisted of unchanged food but no coffee-ground material or bright red blood was noticed. Two weeks prior to admission, the patient became pale and developed heartburn, belching and epigastric pain. Melena persisted following the removal of the medicines from the diet. A review of systems showed that the patient had intermittent attacks of hoarseness following the thyroidectomy. The patient stated that this disappeared after a few days of appropriate therapy and that the difficulty was ascribed to vocal abuse.

The physical examination was essentially negative. Laboratory studies showed an essentially negative urinalysis. On admission, the

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hemoglobin was 38 per cent; the red blood cell count, 2,480,000; the white blood cell count, 9,800 with 75 per cent polymorphonuclears and 24 per cent lymphocytes. By use of transfusions the blood count was raised to 70 per cent hemoglobin with 3,560,000 red cells. Blood sugar was 96 mg. per 100 cc.; blood nonprotein nitrogen, 23 mg. per 100 cc.; total protein 6.09 gm. per 100 cc. and icterus index 13.

Gastro-intestinal roentgenography was done, and the diagnosis of carcinoma of the pylorus was made. A total gastrectomy was performed on March 14, 1946. Anesthesia was carried out by ethylene ether with a Magill endotracheal tube; the patient was under the anesthetic for 3¾ hours. The diagnosis of carcinoma of the stomach was confirmed grossly. The operation was uneventful as was the recovery from the operation. Microscopic sections of the gastric specimen showed adenocarcinoma with secondary ulceration, and the patient was discharged improved on April 2, 1946.

The patient was readmitted on April 13, 1946, complaining of vomiting, abdominal pain which was cramp-like, and diarrhea. Intestinal obstruction was suspected but proved not to be present, and under appropriate therapy the symptoms cleared up, and she was discharged improved on April 17, 1946.

The patient was readmitted on May 9, 1946, and this time her complaint was one of hoarseness which persisted from the time of the gastrectomy on March 14; she stated that she had complained of hoarseness on her second admission but that it was not at that time severe. However, in the past three weeks it had increased in severity, and she had become practically aphonic. Physical examination was essentially negative except for the laryngeal findings.

There were two, large, pedunculated, granulomatous masses apparently attached to the vocal processes and almost completely occluding the posterior half of the larynx; these findings were discovered under indirect laryngoscopy. On May 9, 1946, under local anesthesia, direct laryngoscopy was performed. The above described masses were seen to be about the size of a pea, anterior to the cords at the posterior commissure. These were removed with polyp forceps. Histopathological study demonstrated granulation tissue with organization.

Comment. There are five other cases reported in the literature; Kearney[1] reviewed the literature and added a case of his own. He

found that at least two of the previous cases (Clausen[2] and Cohen[3]) and his own case had in common the fact that the lesion was located in the posterior portion of the larynx. Our case conformed to this anatomical location. In two of the earlier cases, those of Gould[4] and Smiley,[5] the lesions recurred and had to be removed again. Our patient is well one month following excision of the lesion. A significant feature of our case is the fact that the patient had had previous laryngeal difficulty manifested by hoarseness occurring after the thyroidectomy. The patient had been repeatedly examined by one of us by indirect laryngoscopy during this period. On these occasions, only subacute laryngitis was found. Consequently, the production of the lesion can be directly attributed to the use of the endotracheal tube. The previous history of hoarseness confused the issue so that complaints of the patient concerning the hoarseness were minimized.

In view of the possibility of this type of granuloma occurring after the use of an endotracheal tube, one should carefully investigate symptoms referrable to the larynx after intratracheal anesthesia. The probability exists that adequate early therapy may prevent the formation of the polypoid pedunculated lesion.

The duration of anesthesia was somewhat longer in this case than in the other cases in the literature in which the endotracheal tube was used, but this report is not intended to discourage the use of the tube for that length of time inasmuch as so few cases have been reported.

CONCLUSIONS

1. A case is reported, the sixth in the literature, of polypoid granuloma of the larynx following intratracheal anesthesia with the endotracheal tube.

2. The literature is reviewed and produces only five other cases of this lesion.

3. The possibility of the earlier recognition of the cause and effect relationship of the tube to this lesion may prevent the occurrence of the polypoid growth with pedunculation.

Histopathological report by Dr. K. Yardumian, Pathologist at the Montefiore Hospital.

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XX

GRANULOMA OF THE LARYNX

A LATE COMPLICATION OF ENDOTRACHEAL ANESTHESIA

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Recently we have seen two cases of granuloma of the larynx which we feel are secondary to the passage of the endotracheal tube. In reviewing the literature I have found four reports on granuloma of the larynx occurring following endotracheal anesthesia. In 1940 Smiley[1] discussed one case and reviewed three cases previously reported: those of Clausen in 1932,[2] Gould in 1935,[3] and Cohen in 1936.[4] In 1942 Farrior[5] reported a case of contact ulcer of the larynx with exuberant granulation tissue which followed endotracheal anesthesia.

CASE 1.—A 40-year-old housewife was seen whose chief complaint was hoarseness of six weeks' duration. The patient had had a right subtotal hemithyroidectomy for colloid adenomatous goiter performed at another hospital four months previously. Endotracheal anesthesia was given and no abnormalities of the larynx were noted. The postoperative course was uneventful and the patient stated that she was well until about six weeks prior to admission when she noticed her voice becoming hoarse. This increased until, at the time of admission, she was very hoarse and her voice would disappear completely in the middle of a sentence. She had occasional choking episodes. Indirect laryngoscopy revealed a smooth, rounded, dark red mass occupying about one half of the trachea and arising below the right vocal cord just anterior to the vocal process of the arytenoid cartilage. The mass moved on deep respiration and on coughing would push up against the cords.

The mass was visualized by direct laryngoscopy and a specimen was taken for biopsy. This was followed by a moderate amount of bleeding which obscured the operative field. The pathological report was "granulation tissue showing active inflammation." Two weeks later a second direct laryngoscopy was performed and this time the operator grasped the pedicle below the vocal cord. A soft, rounded, pedunculated mass 1 x 1 x .8 cm. was removed. The pathological description was: "The section consists of a small tumor-like mass of fibroblasts and young capillaries which seems to have a

central stalk. This stalk or pedicle is composed of squamous epithelial lined connective tissue. Near the center of the tumor large congested capillaries are present. Nearby are hemosiderin granules of old focal minute hemorrhages. The fibroblastic connective tissue is dense near the center and all fibers seem to radiate out from the central stalk. Near the periphery there is considerable edema and neutrophiles and eosinophiles are present throughout. The outer margins are composed of a ring of fibrin. Diagnosis: granuloma pyogenicum."

The postoperative course was uneventful and the patient regained her normal voice. No recurrence has been seen on subsequent examinations over the three months' period that the patient was available for study.

CASE 2.—A 54-year-old man was admitted to the hospital; his chief complaint was coughing, occasional hemoptysis, and increasing hoarseness. The patient had been operated upon three months previously and a craniopharyngioma was removed under endotracheal anesthesia which was maintained for five hours. The endotracheal tube was passed by direct vision and no abnormalities were noted at that time. The patient was aphasic for four days following operation (intracranial origin) and his voice returned without hoarseness. Two months after the operation the patient developed a cough, noticed that his sputum was occasionally blood-tinged and that his voice was hoarse. He was admitted to the hospital three months after operation and indirect laryngoscopy revealed a smooth, grayish, polypoid mass attached to the upper surface of the posterior third of the right vocal cord and to the vocal process of the arytenoid cartilage.

The mass was visualized by direct laryngoscopy and a specimen was taken for biopsy. The mass, though it did not appear so, was quite vascular and the bleeding obscured the operative field. A second operation will be necessary to remove the remaining granulation tissue. The pathological description was: "The section presents two pieces of tissue one of which is lined by stratified squamous epithelium. The second piece of tissue appears to be continuous with the first but the lining epithelium is missing. In general the section is composed of loose edematous young fibroblasts, among which many capillaries are noted. Neutrophiles are abundant and a few eosinophiles and lymphocytes are present. Diagnosis: acutely inflamed granulation tissue."

Comment. In each of the cases of granuloma of the larynx reported herein there was a history of an operation in which endo-

tracheal anesthesia had been given a few months prior to the onset of the patients' present laryngeal complaints. It would seem that the granuloma had its origin in an ulcer which resulted from an abrasion incurred in the passage of the endotracheal tube. From the location of the tumor it appears that it is most likely to form if the abrasion is on the posterior end of the vocal cord or on or near the vocal process of the arytenoid cartilage where it is subject to trauma in the use of the voice.

CONCLUSIONS

1. A granuloma of the larynx occasionally occurs following the use of endotracheal anesthesia.

2. The larynx should be examined following endotracheal anesthesia and if any abrasions are seen the patient should be placed on voice rest and receive appropriate treatment. This should be followed with periodic examinations until the lesion is healed.

ALBANY HOSPITAL.

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XXI

ACOUSTIC NEURITIS ASSOCIATED WITH KERATITIS

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BOSTON, MASS.

I wish to report three cases of severe bilateral acoustic neuritis associated with interstitial keratitis. In each case this apparent syndrome was characterized by the presence of a nonluetic interstitial keratitis in conjunction with vertigo, tinnitus and deafness. The neuritis terminated after a period of a few months in complete bilateral nerve deafness and loss of vestibular function. The deafness was concomitant with the decreased vestibular response. Although the activity of the keratitis tended to vary from day to day, there was no appreciable change over a period of a year in any of the cases.

Mogan and Baumgartner[1] in 1934 reported the case of a 26-year-old male who, two months after the appearance of a bilateral interstitial keratitis, experienced severe vertigo, nausea, tinnitus and deafness. Seventeen months after the onset, vertigo was still present and serviceable hearing remained. The Wassermann test was negative. A bilateral cervical ganglionectomy was done on the assumption that the inner ear disorder was due to a vasomotor disturbance, causing an extravasation of serum into the scala media. Immediate post-operative results were excellent, with cessation of the keratitis, freedom from vertigo and partial recovery of the hearing. A follow-up report ten years later[2] revealed no further vertigo or loss of hearing and a decrease in tinnitus.

On the basis of this report, a right superior cervical sympathetic ganglionectomy was done on one of our patients early in the course of the disease while serviceable hearing remained. The progress of the disease was not stopped. Nor were we able, with other therapeutic measures, to alter the course of the disease in any of the patients. It may be significant that in all of our patients the disease was much more rapidly progressive than in the case reported by Mogan and Baumgartner. The etiology was not discovered.

CASE 1.—J. L., a 30-year-old, single girl of Italian ancestry was admitted to the Massachusetts Eye and Ear Infirmary on January

From the Department of Otolaryngology of the Massachusetts Eye & Ear Infirmary, Boston, Mass.

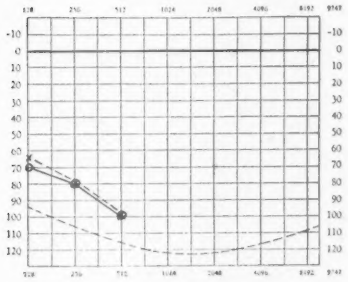

Fig. 1.—Audiogram of Case 1, made Jan. 31, 1944, seven months after the onset of her present illness. (Right ear, O; left ear, X.)

17, 1944. At this time she could not hear the spoken voice and was unable to read because of extreme photophobia and lacrimation. The history was obtained from the mother.

Except for an episode of "ptomaine poisoning" just prior to the present illness, she had always been in good health. Seven months ago she experienced sudden deafness in the left ear, associated with vertigo, tinnitus, nausea and vomiting. This was followed two months later by an exacerbation of the vertigo, with deafness and tinnitus in the right ear. Since its onset the tinnitus increased in severity, while the vertigo gradually subsided. Early in the course of the present illness, she noted irritation of the eyes, and a few days before her hospital admission she had severe photophobia, lacrimation and diminished vision. Her past health had been excellent and there was no history of familial deafness, allergy, or other chronic disease. Wassermann tests on both parents were negative.

Seven months after the onset of the present illness the physical examination was negative except for the eyes and the eighth nerve involvement. There were no findings suggestive of congenital syphilis. The temperature, pulse and respiration were normal.

The ophthalmoscopic examination showed bilateral deep interstitial opacities that were patchy and associated with moderate ciliary injection. There was marked photophobia and lacrimation.

The ear, nose and throat examination was essentially negative. The tonsils were small and atrophic. Massive caloric and Bárány turning tests produced no vestibular response in either ear.

Audiometric responses (Fig. 1) were obtained only after stimulation with the continuous frequency coil, and then only at maximum intensities at three frequencies. Obviously she had a profound bilateral nerve deafness, with no residual hearing in the speech range. Two months later there was an absence of even pure tone response.

The laboratory data were as follows:

Blood Count: Hemoglobin, 94%; red blood count, 4.8 million; white blood count, 12,100; polymorphonuclears, 68; large lymphocytes, 2; small lymphocytes, 18; monocytes, 12. Second Specimen: polymorphonuclears, 62; small lymphocytes, 22; monocytes, 11; eosinophiles, 5.

Urinalysis: Negative.

Serology: Wassermann negative on two tests. Hinton negative on two tests and once in dilution.

Cerebrospinal Fluid: Normal pressure and dynamics; cell count: polymorphonuclears, 1; lymphocytes, 3; protein 25 mg. per 100 cc.; sugar 62 mg. per 100 cc.; colloidal gold, 0001110000; Wassermann test negative; culture negative. On two subsequent examinations, the cell count was 0.

Sedimentation Rate: 0.2 mm. per minute.

Blood Chemistry: Nonprotein nitrogen 22.5 mg. per 100 cc.; sugar 87 mg. per 100 cc.; calcium 9.5 mg. per 100 cc.

X-ray: Both mastoids normal. Chest clear. Sinuses negative except for a cyst in the outer wall of the right antrum. (One week later the sinuses were normal.) Lower right first bicuspid abscessed. (Extracted.)

Culture of Conjunctival Sac (both eyes): No growth.

In spite of a course of sulfadiazine, intravenous typhoid therapy, potassium iodide, large doses of vitamins A, B and C (orally), and local treatment to the eyes, the course of the disease was not materially altered. When last seen, a year later, the interstitial keratitis was still active. She had had no further attacks of vertigo and the deafness and tinnitus remained.

Case 2.—E. C., a 22-year-old, single girl of Italian ancestry, was first seen three months after the onset of her present illness. This began with pain, redness and photophobia in the left eye, and a few days later had similarly affected the right eye. One week later she

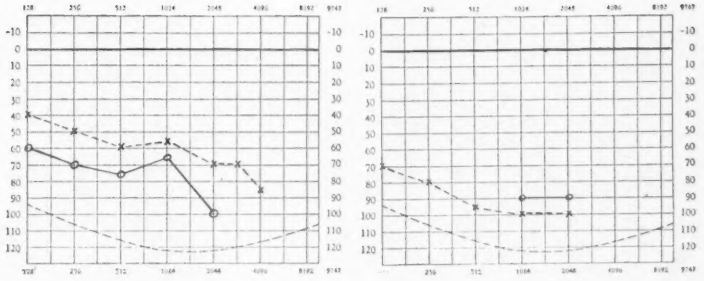

Fig. 2.—Audiogram of Case 2, made Sept. 22, 1944, three months after the onset of symptoms and prior to right superior cervical sympathetic ganglionectomy. (Right ear, O; left ear, X.)

Fig. 3.—Audiogram of Case 2, made Dec. 14, 1944, two months after a right superior cervical sympathetic ganglionectomy. (Right ear, O; left ear, X.)

experienced severe vertigo, tinnitus and deafness. Because of the severity of the vertigo, she was confined to bed for over a month. There was persistent nausea but no vomiting. The initial loss of hearing was sudden, and the tinnitus was described as a "low-pitched roaring." During the three months since the onset, the deafness had progressed and the tinnitus remained severe, while the vertigo had subsided. Subjectively there was no change in the condition of the eyes.

The past medical history was essentially noncontributory. During the past few years the patient had lost 20 pounds and was treated for "anemia" by sodium cacodylate injections. She was not subject to an unusual number of upper respiratory infections. There was no history of food idiosyncrasy or other allergy, and she had taken no medication other than an occasional aspirin tablet. Both parents were living and in good health.

Examination three and one half months after the onset of symptoms was as follows:

The ear, nose and throat examination showed no local pathological changes. The tonsils were absent. There were no palpable cervical glands. Both drums were normal and both eustachian tubes were patent. Massive cold caloric and Bárány rotation tests were indicative of marked vestibular hypofunction.

The audiogram (Fig. 2) showed a loss greater than 50 decibels bilaterally throughout the speech range.

The ophthalmoscopic examination showed vision in the right eye to be 20/70; in the left eye, 20/200. Both eyes showed mild ciliary injection, with patchy interstitial infiltrates deep in the corneal stroma. There were a few cells in the anterior chamber and some interstitial vascularization in left eye but none in the right eye. There were no synechia, and the fundi were normal.

The laboratory data were as follows:

Urinalysis: Negative for albumen and sugar.

Serology: Wassermann test negative twice. Hinton test, in dilution, negative once.

Blood Count: First specimen—Hemoglobin, 93%; red blood count, 5.05 million; white blood count, 12,000; polymorphonuclears, 77; lymphocytes, 24; monocytes, 2; basophiles, 1; eosinophiles, 4. Second specimen: white blood count, 16,300; polymorphonuclears, 72; lymphocytes, 18; monocytes, 8; eosinophiles, 6.

Blood Chemistry: Protein 7.68% (albumin 5.6 and globulin 2.07%, A/G 2.7). Nonprotein nitrogen 24 mg. per 100 cc.; prothrombin time 26 seconds; cholesterol 192 mg. per 100 cc.; phosphate 3.9 mg. per 100 cc.

Cerebrospinal Fluid: Initial pressure (poor relaxation) 170 mm. of water; final pressure 105 mm. of water; cell count 0; protein 36 mg. per 100 cc.; sugar 72 mg. per 100 cc.; colloidal gold 0000110000; Wassermann test negative; culture negative.

Culture of nose: Coagulase negative hemolytic staphylococcus aureus.

Culture of Conjunctival Sac (both eyes): No growth.

X-ray: Chest clear. Both mastoids normal. Spleen and liver of normal size.

On October 28, 1944, four and one half months after the onset of the present illness, a right superior cervical sympathetic ganglionectomy was done on the basis of the case previously reported by Mogan and Baumgartner.[2] However, on December 14, 1944, a reevaluation of the cochlear and the vestibular functions showed

that the progress of the disease had not been interrupted. At this time the deafness (Fig. 3) had progressed so that there was no response to bone conduction in either ear. There was little response to air conduction in the right ear and only a minimal pure tone response in the left ear. Vestibular function was less than indicated on preoperative tests. The tinnitus was less pronounced and the vertigo steadily diminished. There was no essential change in the eyes, although daily variation in the severity of the symptoms had been the rule.

CASE 3.—H. C., a 22-year-old, single girl of Greek ancestry was first seen on February 14, 1944. Six weeks previously she developed marked photophobia, lacrimation, blurring of vision and redness of her eyes. This was followed in two weeks by severe vertigo and tinnitus. The vertigo continued and was accompanied by vomiting, as often as seven or eight times a day. The deafness was overshadowed by other symptoms so that the date of onset is not certain. At the onset of the vestibular symptoms, however, there was associated pain in the left ear, and a myringotomy is said to have been done with no subsequent discharge.

The past medical history revealed no significant organic disease. Both parents and three siblings are living and well.

The physical examination six weeks after the onset of symptoms was negative, except for the local disease.

The ophthalmoscopic examination showed the vision in the right eye to be 20/70. There was moderate ciliary redness along with active interstitial corneal opacities, with many fine deposits on the back surface of the cornea and many cells in the anterior chamber. The vision in the left eye was 20/50. There were few cells in the anterior chamber and a few mutton fat deposits on the posterior surface of the cornea centrally. Some faint opacities in the deep corneal stroma could be seen.

The ear, nose and throat examination showed the right drum intact with normal landmarks. The mobility was normal and the middle ear structures visible. The eustachian tube was patent. The left drum had poor lustre and was slightly retracted with poor mobility. The eustachian tube was patent. The nasal mucous membrane was clean with no evidence of allergy. There was a septal deviation to the left. The sinuses were clear to transillumination.

The nasopharynx was negative and the eustachian tube orifices clear. The tonsils and adenoids had been removed and there was

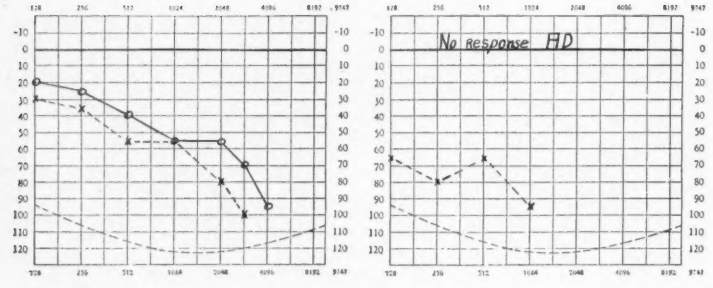

Fig. 4.—Audiogram of Case 3, made Feb. 18, 1944, two months after the onset of symptoms. (Right ear, O; left ear, X.)

Fig. 5.—Audiogram of Case 3, made March 16, 1945, fifteen months after the onset of symptoms. (Right ear, O; left ear, X.)

very little residual lymphoid tissue. The teeth were in excellent repair. The larynx was normal.

Massive caloric tests on each side with water at 40° F. and 68° F. produced no nystagmus, past-pointing, falling or subjective vertigo. Two months after the onset of the present illness an audiogram showed a marked bilateral nerve deafness (Fig. 4). There was one foot of conversational hearing in the right ear and none in the left. The laboratory data were as follows:

Urinalysis: (Three specimens) negative except for albumen on one occasion.

Blood Count: First specimen: white blood count, 18,600; polymorphonuclears, 63; large lymphocytes, 2; small lymphocytes, 26; monocytes, 8; eosinophiles, 1. Second specimen: red blood count, 4.5 million; white blood count, 12,800. Third specimen: red blood count, 4.04 million; hemoglobin, 86%; white blood count, 13,700;polymorphonuclears, 76; large lymphocytes, 2; small lymphocytes, 14; monocytes, 8.

Serology: Blood Hinton tests negative three times and once in dilution.

Cerebrospinal Fluid: Initial pressure 170 mm. of water (poor relaxation); fluid clear; protein 29 mg. per 100 cc.;

sugar 63 mg. per 100 cc.; cell count, 0; colloidal gold, 0000000000.

Culture of Conjunctival Sac: Coagulase positive hemolytic staphylococcus aureus.

X-rays: Skull, mastoids and chest normal.

The temperature, pulse and respiration were normal. She was given sedatives, local treatment to the eyes, vitamin B, potassium chloride and intramuscular injections of bismuth subsalicylate, with no relief. During the next three months after her discharge, the vertigo became less severe, the deafness progressed and the nystagmus disappeared. The eyes showed considerable variation from day to day but remained essentially unchanged. Four months after the onset of the present illness, she was again admitted for study. No serviceable hearing remained and there was no vestibular response. Vision in the left eye was 20/100; in the right eye 20/70. The keratitis had not improved. Repeat laboratory studies, including Hinton, Kahn and Wassermann serologic tests, were normal.

She was again seen on March 16, 1945 (15 months after the onset of the present illness). At this time, there was practically no auditory (Fig. 5) or vestibular response, no vertigo and no tinnitus. The eyes were white and quiet. In the posterior portion of the central cornea there were many highly reflective areas accompanied by extensive vascularization. There were a few pearly white deposits on the posterior surface of the cornea which did not look active. The picture was essentially the same for the two eyes.

DISCUSSION

The otological literature abounds with cases of acoustic neuritis due to a variety of causes. Johnson[3] has recently reviewed these. Many more, in which the etiology has not been determined, have been presented. The striking similarity of these three cases of acoustic neuritis with interstitial keratitis, in addition to the one previously reported case,[1] suggests a well-defined clinical syndrome. In each of these the onset was characterized by violent vertiginous attacks with deafness and tinnitus, in conjunction with the abrupt appearance of bilateral interstitial keratitis. The disease was initiated by ocular signs in Cases 2 and 3 and by signs of eighth nerve involvement in Case 1. The longest interval of time between the ocular and the acoustic nerve manifestation was one month (in Case 1).

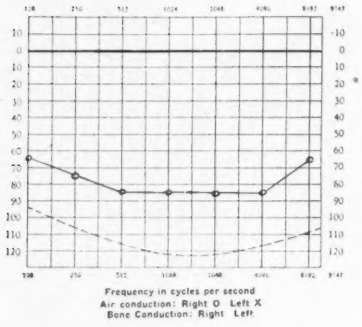

Fig. 6.—Audiometric curve showing the hearing loss at which it may still be possible to understand speech. (From Bunch, C. C., Clinical Audiometry,[4] C. V. Mosby Co., with permission.)

The disease was consistently bilateral throughout its course and progressed to total loss of serviceable hearing.

Bunch[4] has plotted an audiometric curve showing the hearing loss at which it may still be possible to understand speech (Fig. 6). On the basis of this curve the amount of residual hearing was, in all three of our cases, insufficient to enable these patients to understand speech even with the use of the most powerful vacuum tube hearing aids.

Although the keratitis varied in severity from day to day, it ran a characteristically chronic course, even after cessation of vestibulo-auditory symptoms.

With rare exceptions, interstitial keratitis is caused by syphilis. The accompanying deafness makes it all the more necessary to rule out syphilis as the etiological factor in these cases. Dr. David L. Cogan of the Department of Ophthalmology, having observed the ocular phase of the disease in these patients for over a year, has concluded that the keratitis differed markedly, both in onset and course, from that of syphilitic interstitial keratitis.

Congenital luetic deafness is commonly described as occurring most frequently between the tenth and twentieth years, having a higher incidence in females, and being bilateral. It is not uncommon for it to progress rapidly, terminating in severe perception deafness with loss or disturbance of vestibular function. Vestibular tests

may show peculiar and inexplicable reactions, such as lack of response to the Bárány turning test in the presence of a reaction to caloric stimulation.[5] None of our cases showed any of these bizarre reactions to vestibular stimulation. Decrease in activity of the static labyrinth closely paralleled the loss of cochlear response. While the deafness, then, is not unlike that occasionally seen as a manifestation of congenital lues, its relationship to the interstitial keratitis does not conform to the usual pattern. Dalsgaard-Nielsen[6] studied 173 patients with a past history of syphilitic interstitial keratitis, the average period of observation being 20 years. Twenty-seven, or 15.6 per cent, of these patients had a loss of hearing. The severity of the deafness is not explained, but it is of significance that he mentions no cases of total loss of serviceable hearing. Nor did vertigo appear to be a predominant symptom. In his cases, however, the deafness always developed later than the keratitis, the interval between keratitis and deafness varying from one to 31 years. He states, furthermore, that the deafness appears preferably in patients whose keratitis, unlike those herein reported, has taken a rather malignant course, and in whom other syphilitic manifestations appeared.

Neither the type of deafness, the keratitis, nor their time relationship to one another, is, however, sufficient to exclude the possibility of syphilis. Toward the elimination of this etiological factor, repeated serologic tests on these patients, and in two cases upon their parents, were conducted with negative results. Jeans and Cooke[7] in studying 193 cases of syphilitic interstitial keratitis found that 187, or 97.9 per cent, had a positive Wassermann test. They concluded that in the vast majority of cases of syphilitic interstitial keratitis, the Wassermann reaction is strongly positive and tends to remain so during long periods of intensive treatment.

Inquiry into the family histories gave no evidence of abortion, still births, or congenital syphilis in any of the siblings. Physical examination, with a search for other characteristic stigmata, was in each case negative. Thus there was no sound basis for a diagnosis of congenital syphilis in any of these patients.

Steegman[8], in discussing neuronitis of the cranial nerves, frequently refers to involvement of the eighth nerve. Neuronitis, he believes, is due to a virus infection of the nerve itself in which the clinical picture follows a routine pattern. There is an initial adjacent infection, followed by a period of latency, and then symptoms of cranial nerve involvement. In his cases recovery seemed to be

the rule, and he presents no cases with complete loss of eighth nerve function. In our cases examination was not made to determine the presence of a virus infection. But all three patients, at some time during their hospitalization, had a slight leukocytosis, and two of them had a mild eosinophilia.

All three patients were women in the young adult age group, with no manifestations of allergy, dietary deficiency, or other chronic disease. No history of recent infection, trauma or medication was obtained.

Treatment was rendered somewhat difficult for two obvious reasons. In the first place, any therapy undertaken in a perception deafness, particularly when the cause is not clearly defined, is of necessity empirical. Secondly, the degree of deafness and the loss of vestibular function which were present when these patients were first seen, suggest that irreversible changes had already occurred in the end organs. All foci of infection were removed in so far as possible. A course of sulfadiazine was given in Case 1. Cases 1 and 3 were not benefitted by large doses of vitamin B complex, although it was given orally rather than intravenously as recommended by Brandenberg.[9] A course of intravenous typhoid injections given primarily for the keratitis in Case 1 was without effect. Potassium chloride and a series of intramuscular injections of bismuth subsalicylate did not influence the course of the disease. A right superior cervical sympathetic ganglionectomy, done in Case 2, while serviceable hearing still remained, did not interrupt the progress of the neuritis.

SUMMARY

Three cases are presented of a syndrome consisting of an eighth nerve neuritis, in conjunction with interstitial keratitis. Both the keratitis and the acoustic neuritis, as well as the time relationship between them, differed considerably from that of congenital syphilis. The disease progressed over a period of six to seven months to a complete bilateral loss of function in the acoustic and the static labyrinths. The etiology was not determined, and the possibility of a virus infection was not eliminated. Superior cervical sympathetic ganglionectomy, done early in the course of the disease on one patient, was without benefit.

243 CHARLES STREET.

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XXII

PERICHONDRITIS OF THE LARYNX SECONDARY TO INTERSTITIAL APPLICATION OF RADIUM TO THE THYROID GLAND

REPORT OF A FATAL CASE

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DANVILLE, VA.

The danger of necrosis of the laryngeal cartilages following roentgen ray or radium therapy to the thyroid gland has been pointed out in the past. A fatal case of perichondritis of the larynx following the interstitial application of radium to the thyroid gland is reported to reemphasize the hazard of this form of treatment.

Kaplan[1] states, "Cartilage, because of its relative avascularity and poor recuperative powers, is readily destroyed by technical errors in radium therapy . . . Tracheal cartilage necrosis has too often caused the painful death of patients who were incorrectly treated by having radium directly implanted to its vicinity. In some instances local surface application could have prevented necrotic results had they been used instead of interstitial irradiation."

In the discussion of a paper on osteoradionecrosis, Clerf[2] mentions two cases of irradiation necrosis of laryngeal and tracheal cartilages following radiation therapy for exophthalmic goitre; the symptoms started five and seven years after treatment. One patient died and the other survived with a large unhealed area over the front of the neck. After tracheotomy for laryngeal stenosis in the latter case the wound broke down, with much loss of soft tissue as well as the thyroid, cricoid, hyoid, and tracheal cartilages. In the same discussion, Tucker[3] reported a case of extensive extrinsic carcinoma of the larynx, treated with intralaryngeal radium and external roentgen therapy. The patient lost all of the cartilaginous structure of the larynx. Tucker pointed out the important fact of great variation in individual tolerance to irradiation. He believes that radiation therapy should be limited to cases of extrinsic carcinoma of the larynx.

In the radiation treatment of carcinoma of the larynx, perichondritis is recognized as a justifiable complication in the treatment

of a disease potentially fatal. The serious sequelae of extensive necrosis, sequestration of the laryngeal cartilages, laryngeal stenosis, and asphyxia can be avoided by expert radiological management.

Cutler[4] records two deaths as a direct result of radionecrosis in a series of 118 cases of cancer of the larynx treated with concentrated radiotherapy. One patient died of laryngeal hemorrhage eleven months after treatment. The other died of laryngeal necrosis nine months after treatment. In seven other patients who died, varying degrees of necrosis were present. All seven had far advanced carcinoma of the larynx. Cutler emphasized the fact that the general condition of the patient and the presence of other debilitating diseases lower the tolerance to irradiation. The presence of infection can now be controlled by the sulfonamides and penicillin and they will be of value in controlling the complications of irradiation.

To avoid the disastrous effect of radiation injury to the laryngeal cartilages in inoperable cases of cancer of the throat, Arbuckle[5] advised preliminary removal of the entire thyroid cartilage.

Laryngeal perichondritis is not an uncommon disease. It occurs after direct trauma and may be secondary to certain acute and chronic infections; it may result from extension of inflammation of the oral cavity, it may occur after the use of nasal feeding tubes, and may follow irradiation to the region of the larynx. The diagnosis is made from the history, by the appearance of the larynx, and by palpation. X-ray examination is important. The sequelae, stenosis and atresia of the larynx, chondrial necrosis, and asphyxia are most serious. The stenosis is most severe when caused by irradiation.

REPORT OF A CASE

Mrs. A. J. F., 32-year-old white woman, was first admitted to the Memorial Hospital on February 27, 1944. She complained of nervousness and loss of weight. A diagnosis of toxic adenoma of the thyroid gland was made. For unrecorded reasons interstitial application of radium needles was advised. The basal metabolic rate was not noted. On February 28, 1944, through a necklace skin incision six ten-milligram radium needles were implanted into the thyroid gland and left in place 45 hours, a total dose of 2700 mg. hours. She was discharged on March 4, 1944.

I first saw the patient on her second admission to the hospital on November 1, 1944, eight months after the radium application. She complained of hoarseness, slight difficulty in breathing, a pro-

ductive cough, and severe pain on the right side of her neck, radiating to her right ear. She stated that she had improved since the radium treatment, and generally felt better until the present symptoms started the month before. Physical examination showed a slender underweight woman with slight exophthalmos. The skin over the thyroid gland was thickened and markedly erythematous. The structures beneath were immovable and indurated but not tender to palpation. Direct and indirect laryngoscopic examination showed atrophic, reddened vocal cords; the subglottic area was edematous. The trachea contained a purulent exudate. The laboratory examination showed no departure from the normal. X-ray examination of the chest was negative. During her stay in the hospital her pulse rate remained in proportion to the elevated temperature, which varied from 99° to 100° F. daily. Under conservative treatment, hot compresses to her neck, benzoin inhalations, and analgesics, the patient improved and requested her discharge on November 4, 1944.

She returned to the hospital two weeks later complaining of increased difficulty in breathing and hoarseness. The physical examination was the same as on the previous admission except for moderate obstructive dyspnea and a marked increase in the size of the erythema of the lower portion of the neck; the erythema now extended down onto the anterior chest wall.

A tracheotomy was suggested but was deferred when further observation appeared to indicate a lessening of the dyspnea. The morning of the second hospital day the patient stated she had slept better than she had in several weeks. Three hours later her difficulty in breathing suddenly increased, and she died before the completion of an emergency tracheotomy.

At operation the incision was made as low as possible to avoid the indurated immobile tissues over the thyroid gland. Beneath the skin and the subcutaneous layer of scar tissue, the tissues were found to be necrotic, filled with small abscesses and foul smelling pus. The tracheal rings were soft; the tracheal lumen was dilated and filled with purulent material. After death the larynx was removed.

Gross examination of the larynx showed posteriorly on the right side a large erosion of the lamina of the cricoid cartilage and the upper tracheal cartilages. This area, the size of a dime, was covered by a necrotic membrane. The microscopic examination of the larynx was done through the courtesy of the Army Medical Museum and was reported as follows:

"Section taken through the area of the gross necrosis in the cricoid cartilage shows an infiltrate of polymorphonuclear leukocytes surrounding acellular debris. Outside this, there is a zone of dense fibrosis in which one sees the changes usually associated with radiation reaction. There are distorted fibroblasts, some of them showing huge vacuoles in their nuclei and others with bizarre nuclei which suggest ganglion cells. The changes in the vessels are suggestive of radiation reaction. Some arterioles are completely obliterated, others show subendothelial thickening and hyalinization. Dilated capillaries are particularly evident in the submucosa. The surface of the cricoid cartilage near the ulceration is necrotic and the deeper portions show an acidophilic staining matrix in which the cell spaces are empty or contain red cells or degenerated cartilage cells. The mucus glands show some dilatation and a great deal of surrounding inflammatory infiltrate but this change, of course, would be seen in any area of necrosis and infection. The dense fibrosis and atrophy of the alveoli seen in the thyroid gland one would expect after irradiation but they are also the changes commonly seen in nodular goitre. None of these findings is specific for radiation effect. The most significant, in our opinion, are the peculiar fibroblasts, the hyalinization of arterioles and the ectatic capillaries."

Comment. In the early days of radiation therapy, injury to the laryngeal cartilage was not uncommon. Severe and sometimes fatal cases of radionecrosis of the laryngeal cartilages were repeatedly reported in the literature. Improved technique has reduced markedly the incidence of this complication. It is apparent that there is an individual variation in the tolerance of the cartilage to injury.

In the case reported there was an interval of seven months between the radium therapy and the onset of the laryngeal symptoms; tissue damage has been known to occur as long as seven years after irradiation. In this case the cricoid cartilage suffered the most damage. Jackson states that the cricoid is the most frequently and disasterously affected of all the cartilages. The pre-irradiation removal of the thyroid cartilage would then seem to be insufficient.

CONCLUSION

A fatal case of perichondritis of the larynx secondary to the interstitial application of radium to the thyroid gland is reported. Modern methods of irradiation to the neck have reduced this complication to a minimum. Radionecrosis of the laryngeal cartilages, however, will be an occasional complication and, because of its seriousness, deserves close observation and prompt treatment.

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XXIII

CONGENITAL CYSTS OF THE NASAL BRIDGE

BERT E. ELLIS, M.D.

INDIANAPOLIS, IND.

During the past year a man was referred to me who had an unusual tumor mass on the bridge of his nose. This was a condition I had not encountered before, and after making inquiry I found only one other rhinologist in our local group who had seen a similar case. Upon investigating the literature I found only a few cases reported and only two in this country previous to the outstanding report by New[6] in 1937 in which he reported 13 cases. Examination of the records of Indiana University showed only one case of dermoid cyst of the nose reported, one of the eyelid and five of the forehead. In view of the rarity of the condition I felt that a review of some of the other cases along with the presentation of this one might be worth while.

REPORT OF A CASE

This man was a white male, aged 25, who appeared to be in good health except for the large fluctuating mass on the bridge of his nose. Below the mass toward the tip of the nose was a dimple in the skin from which grew five or six soft hairs.

His history was irrelevant except with regard to his nose. He had been born with a noticeable hump on the bridge of his nose and an occasional drop of discharge had exuded from the dimple at the tip of his nose until two years ago when an abscess formed in the mass on the bridge of the nose. This abscess had been lanced four times during the past two years and evidently contained pus when first seen by me. The referring doctor had inserted a small silver wire into the dimple and found that it slipped into a cavity beneath the nasal bones.

On his first visit this cyst was lanced again and considerable pus evacuated. It was found that a probe could be inserted to the depth of one inch into a cavity beneath the bridge of the nose. There was

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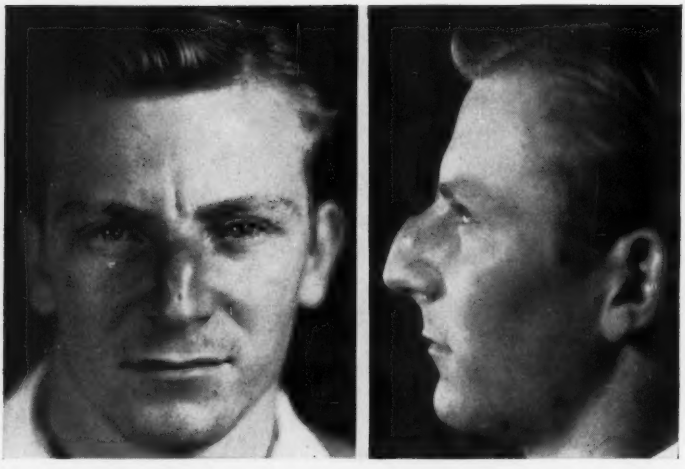

Fig. 1.—Front view before operation.

Fig. 2.—Side view before operation.

also a subcutaneous stalk extending down to the dimple near the tip of the nose. The vertical incision was extended to the dimple and the stalk was resected. Further examination of the cavity beneath the nasal bones showed it to be a bone-walled cavity, somewhat elliptical in shape and about one inch in its longest dimension. The walls were smooth and shining and any efforts to curette the walls were not satisfactory because the lining membrane seemed very thin and adherent. Every effort was made to destroy whatever lining there was to the cavity and the stalk was entirely removed, after which drainage was established into the nose.

The wound healed promptly but six weeks later another abscess appeared. Once more this was drained and a few weeks later a submucous resection was done. This time the cavity was approached from below and the bony walls removed. The hump was sawed down and the lateral walls broken in after an osteotomy was done.

When the osteotomy was done the nasal bridge seemed to break into small pieces, because of the thinned out walls, and did not make a good support for the nose.

To date there has been no recurrence of the abscess and I feel sure that a cure may be expected.

There was no caseous material and no hair found in this cyst, but I believe it must be classified as a dermoid cyst. The fact that this condition had existed from birth and had discharged mucoid material from the dimple on the nose, along with the fact that hairs were growing from the orifice of the cyst, indicates a dermoid cyst. The absence of caseous material and hair may have been due to the fact that this material had been extruded when the abscess had been incised.

Comment. In reviewing the literature on this subject I was impressed by the similarity of the reports. They all seemed to say the same thing. This, no doubt, is due to the similarity of origin of these tumors and to the scarcity of literature to review. It seems to be agreed that these tumors arise from sequestration of surface epithelium during embryonal life.

In the early embryo the face is represented by an opening from which five fissures radiate—the upper orbitonasal fissures, two lower mandibular fissures and an intermandibular one. These fissures afford many chances for embryonic imperfections such as persistence of the fissure, imperfect closure of the fissure and sequestration of surface epithelium. It will be seen that such tumors as dermoids must arise along the line of these embryonic fissures. Therefore, dermoid cysts at the external and internal angles of the orbit (orbitonasal sulcus) are not extremely uncommon.

It should be remembered that some of these tumors at the inner angle of the orbit penetrate the bone and become continuous with the dura.

Dermoid cysts of the bridge of the nose are much less common than those around the orbit. In New's report the nasal dermoids represented 2.6 per cent of the 103 dermoids found around the head and neck. Nine of the cysts in his cases were in the bridge of the nose, one at the tip, one at the base of the columella and one in the septum. Since there is no embryonic fissure proper in the midline of the bony part of the nose, it must be assumed that they arise by the same process as cranial dermoids.

Luongo[2] gives us the following explanation of the formation of these nasal midline dermoids: "In the skull of an early embryo the fronto-nasal plate, which ultimately forms the nose, consists of a lamina of hyalin cartilage covered externally with skin and internally

with mucous membrane. After the third month sections made through the nasal capsule immediately anterior to the ethmoid show that the skin is being dissociated from the underlying cartilage by bony tissue which eventually becomes the nasal bones. Ultimately the cartilage disappears as a result of the pressure exercised by these bones. It is reasonable to believe that in the gradual separation of the skin from the cartilage of the frontonasal plate by the intrusion of the nasal bones, small portions of skin or epithelium become sequestrated and eventually develop into dermoids".

The skin-lined recesses in the tip of the nose may be explained, of course, by the fusion of the two globular processes at the internasal fissure, but since these globular processes give rise to a portion of the alae and premaxillae only, the internasal fissure does not extend high enough on the nose to explain the cysts in the bony part of the bridge.

These cysts are much more common in men than in women and embryonically are comparable to hair-lined pilonidal cysts.

The ideal treatment is complete excision but this is not easy in such cases as the one reported in which the cyst extends far under the nasal bones and is so firmly attached to the bone that complete destruction of the lining membrane is a difficult problem. New suggests light cauterization in such cases, avoiding a current strong enough to cause sequestration of the underlying bone. Where the bony part of the cyst is largely in the septum it may be removed from below via a submucous resection.

Hart[5] reported the first case of a dermoid cyst of the nose in this country (1927). At that time he reported 17 cases in foreign countries. Since then Dr. Luongo[2] has reported a case. New reported 13 nasal dermoid cysts in 1937. It will be noticed that this number is more than half as many as have been reported in all the literature elsewhere. I have no doubt that many of these nasal cysts have not been reported but at least they are rare and a better understanding of their pathology will be an aid in the care of such cases.

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XXIV

TEMPORARY COMPLETE PARALYSIS OF BOTH RECURRENT LARYNGEAL NERVES DUE TO AN EXTENSION CAST APPLIED FOR SCOLIOSIS

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NEW YORK, N. Y.

Because the recurrent nerve is especially vulnerable, many cases are encountered in which temporary or permanent paralysis occurs as a result of pressure on it. This condition may develop after an operation on the thyroid gland, when the nerve may be caught in a hemostat, or it may develop as a result of a hematoma occurring after an operation, or it may be due to mediastinal tumors, enlarged lymph glands, or an enlarged heart. There are also many cases in which no cause is apparent. Stretching of the nerve may also damage it, although the amount of such damage is not known, and the authors do not agree on the subject. For instance, Lahey denies its damaging influences, while Heyd takes the opposite view. In the case presented here, external pressure combined with a stretching of the neck caused the paralysis of both vocal cords. No similar case has been found in the literature.

D. D., a white man aged 23, was admitted to the New York Post-Graduate Hospital on March 28, 1946, to have an extension plaster cast applied to correct scoliosis. Five years earlier he had rheumatic fever and polyarthritis with cardiac involvement. Since then the patient noticed an increased humping of the back.

The patient was small and appeared to be poorly nourished. The examination of the chest disclosed the following conditions: The heart was somewhat enlarged; the apex beat was strongly palpable with the suggestion of a thrill; the rate was 110 with an occasional pause of rhythm; and there was reduplication of the first sound at the aortic area. The lungs were normal. The spine showed slight dorsal scoliosis with convexity to the right and moderate dorsal kyphosis from the first to the twelfth thoracic vertebra, with a limitation of motion.

From the Nose and Throat Department of the New York Post-Graduate Hospital and Medical School.

The cast was applied to the trunk, shoulders, neck and head, on March 29, without anesthesia. On March 30 the patient complained that the cast was hurting his ear and was in general very uncomfortable; he was unable to take any solid food; he could not pass urine and had to be catheterized. On April 1 the cast was cut and trimmed by the operating surgeon. On April 2, at 2:00 a. m. the patient complained of a sore throat, difficulty in swallowing, and stated, "the cast hurts my Adam's apple." He felt nauseated and vomited twice. In the morning the cast was adjusted again and cut out at the neck by the surgeon. On April 3, the patient coughed a great deal, had difficulty in swallowing, and coughed up a small amount of red blood. The resident found that the throat was injected and was too sore for the patient to be able to swallow food. Therefore, an infusion of 2,000 cc. of sodium chloride solution was given as well as saline throat irrigations and sulfadiazine by mouth. From April 5 to 9 the patient had increased soreness of the throat, had difficulty even in swallowing liquids, and had occasional choking sensations.

On April 9, early in the morning, the cast buckled at the bottom. During the night, because of the pressure of the cast on the chest, the patient had such difficulty in breathing that he became cyanotic. The cast was removed by the surgeon the following morning but the patient's condition did not improve and resident on nose and throat service requested an emergency consultation. I advised the resident to administer oxygen until I could see the patient. When I saw the patient, who had a severe inspiratory dyspnea, although the oxygen tube in his nose made breathing somewhat easier, his condition was not alarming. Examination of the neck indicated that the larynx was swollen and tender. The color of the throat was normal. The examination of the larynx was made difficult by the external swelling and the forward drop of the epiglottis. Exposure of the glottis showed that both vocal cords were in the midline and that they did not move. The opening of the glottis was no larger than 1 to 1.5 mm. There was no edema of the vocal cords or of the glottis. The diagnosis was paralysis of both recurrent nerves due to the pressure of the cast.

The patient's temperature by this time was 102.6° F. The blood count showed 18,950 leukocytes, of which 87 per cent were polymorphonuclear leukocytes, 7 per cent were lymphocytes, and 6 per cent were monocytes. The surgeon prescribed penicillin, 50,000 units at first, and 30,000 units every three hours thereafter. The patient's breathing was somewhat easier as a result of the constant

administration of oxygen, but he was unable to swallow liquids, was hardly able to speak, had a productive cough, and was very restless throughout the night. A roentgenogram of the chest showed that there was no involvement of the lungs. On the morning of April 10, when the patient's condition remained unchanged, I decided to perform a tracheotomy.

A low tracheotomy was performed under local anesthesia. After the skin incision was made, it was apparent that the tissues around the neck were marked by a hemorrhagic diffusion and were swollen. After separation of the sternohyoid muscle the isthmus of the thyroid almost reached the sternal notch because of the marked swelling. The isthmus was lifted up, after which the trachea was exposed and a No. 6 tube was inserted without difficulty. The mucous membrane of the trachea was a normal color. The patient was returned to bed in good condition.

On April 11 the patient's condition was somewhat improved. The outside dressing and the inside tube were changed. The patient complained of a constant cough which brought up a great deal of mucus. On April 12, although the tube had been changed, the patient complained of pressure on the chest. Therefore, a No. 5 tube was inserted, which did not irritate the patient. However, his temperature rose that night to 104.4° F. and the next day to 105°, notwithstanding the constant administration of penicillin.

Medical examination showed a right lower lobe involvement with dullness and crepitant râles. The diagnosis was lobar pneumonia. The penicillin was increased to 50,000 units every three hours. A roentgenogram of the chest showed density toward the base of the right lobe. The patient was put in an oxygen tent and, in addition to penicillin, sulfadiazine, 2.5 gm. as a first dose, followed by 1 gm. every four hours thereafter, was given along with bicarbonate of soda. On April 14, because the patient's condition remained precarious he was given 500 cc. of whole blood in addition to a continuous infusion which had been administered for several days. Toward evening the patient's temperature began to drop; by the morning of April 15 his temperature was normal and remained so. The blood count was normal. The patient felt much better and was cheerful, and on the following day he was able to eat and talk. On April 16 an examination of the larynx showed that there was some movement on the right vocal cord. The tracheal tube was corked for the day and no discomfort to the patient resulted. On April 18 he got out of bed. The laryngeal examination dis-

closed that there was good movement of the right vocal cord but sluggish movement on the left side. The cork was kept in for 24 hours. The patient slept and ate well. On April 19 the tube was removed and the wound was closed only with adhesive tape. The patient left the hospital on April 20. Ten days later when the outside wound was closed completely, the movements of the cords were normal.

X-ray study of the larynx did not disclose any pathologic condition except a soft tissue swelling.

COMMENT

The complete recovery of the patient can be attributed to two facts. First, the timely removal of the cast relieved the nerve from the combined pressure and stretching and gave it a chance to reestablish its function. Second, while the pressure was uniform and of comparatively long standing, it was not localized on any special point on the nerve. It is also interesting to note that lobar pneumonia developed in spite of the administration of large doses of penicillin and sulfathiazole, but responded promptly to sulfadiazine.

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XXV

OSTEOMYELITIS OF THE JAW—SIMULATING SARCOMA, MYOSITIS OSSIFICANS, AND ACTINOMYCOSIS

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A 15-year-old high school boy was referred on account of a painful swollen right jaw and difficulty in opening his mouth. The previous summer he had been struck a severe blow with a stick. This was followed by severe pain for several weeks. Not until ten months later did his present symptoms develop. He recalled receiving several sharp blows on the jaw while playing basketball. He also admitted the habit of chewing grass straws while working on the farm.

Except for this swollen jaw, the boy gave the appearance of health. He and his aunt stated that the onset of the recent swelling had been mild and was not accompanied by high fever. He had continued to attend school and to play basketball. He had lost no weight and did not give the appearance of having been acutely ill. Following the injury, he had been given an intensive course of sulfanilimide therapy and repeated x-ray treatments.

The conspicuous findings were a firm, rapidly growing, nonsensitive mass, about the size of one half of a lemon, attached to the external surface of the right lower jaw near the angle; there was limitation of motion. The differential diagnosis suggested sarcoma, actinomycosis, osteomyelitis and myositis ossificans.

The usual order of diagnostic investigation was carried out. Cancer was the first consideration. Unfortunately, the roentgen films and the biopsy studies only added to the confusion of this diagnosis.

When the biopsy was done, sarcoma was so definitely accepted as the probable diagnosis that sufficient tissue was not removed. An external periosteal sarcoma seemed to fit the clinical diagnosis. There was a tendency for the tumor to grow away from the bone. Encap-

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Presented before the Staff of the Menorah Hospital, Kansas City, Mo., June 10, 1946.

sulation was incomplete and the tissue was vascular but firm. The roentgenogram was distinguished by practically no tendency to destruction of the cortex and the marrow cavity. Surrounding the bone was the encircling tumor mass which was generally rather opaque.

Opposed to this diagnosis, but at the time completely ignored, is the important statistical information that although osteogenetic sarcomas are the main group of primary malignant tumors found in the jaw bones, only one bone sarcoma is present in 75,000 to 100,000 people, and that only three per cent of the whole group occur in the jaw bones. Thus, osteogenetic sarcomas of the jaw bones are very rare.[1]

Our eagerness to diagnose and report an unusual condition often confuses us and we lose sight of the most valuable diagnostic assistance in our difficult cases, which is to abide by the law of averages and to be very reluctant in selecting for a diagnosis in any confusing case a disease which is extremely rare.

The clinical course of this lesion was in keeping with another lesion, namely, myositis ossificans. Myositis ossificans may follow a severe injury to the muscle which is accompanied by hemorrhage, with ossification springing from the periosteum and spreading along the fascia or tendon. At first, there is an increase of fibrous tissue, chondrification and ossification. Accompanying this is an atrophy of the muscle fibers.

Unfortunately, after securing sufficient clinical evidence to support this diagnosis, the roentgen film, which is so reliable in the diagnosis of myositis ossificans, failed to confirm it.

Three weeks following the first biopsy, which was done under the direct observation of the pathologist, the boy returned for further study. Except for some moderate drainage, which occurred one week after his return, his condition was unchanged.

The wound at the site of the biopsy specimen had healed. There was no apparent enlargement of the mass, and no palpable submaxillary glands were present. He still looked well and, except for difficulty in opening his mouth, expressed no particular discomfort. In fact, his only complaint was that he was obliged to refrain from playing basketball.

This remark, concerning trauma to his jaw, elicited and emphasized the fact that he had had several severe blows to his jaw.

More roentgen films gave no additional information. At this time, however, actinomycosis was considered. The boy again admitted that in the summer he had the habit of chewing grass, which was confirmed by his aunt who stated that she had repeatedly corrected him about this habit.

There was no evidence of any mucous membrane lesion in his mouth, nor had he had any teeth extracted. Clinically, however, this diagnosis fit the lesion perfectly, in that he had a slowly spreading tumefaction, which after several months broke down and ruptured. Following this a more or less permanent sinus persisted. Ankylosis of the jaw gradually occurred, caused by the overproduction of fibrous tissue. Pain was not severe and was often completely absent, the whole picture being one of chronicity.

Considerable misunderstanding has arisen concerning the pathogenesis of this disease. Wright,[2] who is probably the leading authority on this subject, differentiates the bovine and human types of actinomycosis, in that each has different biologic characteristics. Lord[1] has proven by intraperitoneal injections into guinea pigs, from the scrapings of carious teeth, tongues, and crypts of tonsils, that the organism is normally present in the mouth and gastro-intestinal tract of the human. However, against the scientific evidence remains the clinical fact that farmers are more frequently affected.

Actinomycosis should be suspected where there has been a slowly developing, painless, inflammatory process about the face or neck. Often, a fresh abscess is not present when the patient is first seen, and it may be necessary to keep the patient under observation to prove or disprove the presence of the organism.

When we made an opening into the jaw through this firm tumor mass, collections of "sulfur granules" were thought to have been seen. Microscopic examination proved this to be erroneous and when the bone was reached, an entirely different finding was made.

Osteomyelitis, although considered last in this discussion, was given primary consideration and was only discarded because of the absence of certain well-established clinical factors, namely, the mild onset, the absence of chills, extremely high fever, and severe pain. At no place in his history was there any evidence of the stormy onset which is so characteristic of osteomyelitis. Also, the duration of the disease without extension of the infection or the production of a spontaneous fistula or soft tissue necrosis seemed to rule out the diagnosis. However, at this second operation, which was performed

both for the removal of a biopsy specimen and also for complete surgical drainage, the diagnosis was established.

Beneath a thin shell of necrotic cortex, a fairly large cavity filled with necrotic debris was found in the bone. This cavity was "saucered" out, and the overhanging edges removed. Necrotic bone was thoroughly removed and drainage was established. Marine sponge packs were used to keep the wound open and to maintain drainage. His recovery was uneventful. Later it was learned that this boy had received massive doses of sulfonamides at the onset of the disease. Obviously this confused the early normal clinical picture of osteomyelitis.

Although enough time had elapsed from the onset of the disease for sufficient bone changes, such as sequestra, to exhibit themselves in the roentgenogram, yet in this particular area none was present, and it must be kept in mind that sequestration about the jaw does not always occur.

Often there is a slow absorption of bone with an almost equal replacement so that the roentgenogram gives only a hazy moth-eaten appearance. The extensive blood supply to this area probably explains this phenomenon, and the heavy short overlying muscles prevent the usual external evidence of localizing tender points with subsequent necrosis and suppuration. The delayed surgical interference was in keeping with the proper treatment of this disease in this area.

SUMMARY AND CONCLUSIONS

In retrospect, the question arises, why was this disease so difficult to diagnose and why did so many confusing and plausible diagnoses enter into it?

My first impression was that the patient had a sarcoma. The immediate and primary effort was to prove or disprove this diagnosis. The true nature of the condition was not revealed in the repeated roentgen films.

He had received x-ray treatment of an undetermined amount. The large doses of sulfonamides which he had received masked the normal violent primary reaction of the osteomyelitis and no doubt delayed the suppurative processes within and about the infected bone. Repeatedly, I have seen the same confusing and delayed reaction in the mastoid process. Regardless of the acknowledged beneficial effect of the sulfonamides and penicillin, it is unfair to expect such a miracle as the removal of infected bone debris. Eventually, this

unremoved infected foreign body acts as a low-grade secondary focus, which continues to delay healing, often produces secondary complications, and must eventually be removed.

A malignant growth in this area is such a tragedy that it often warps our diagnostic judgment, and we are inclined to suspect it even when it is not present. Actinomycosis and myositis ossificans fit into certain phases of the clinical findings and had to be considered in the differential diagnosis.

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Society Proceedings

SIXTY-SEVENTH ANNUAL MEETING

OF THE

AMERICAN LARYNGOLOGICAL ASSOCIATION

Chicago, Ill., May 29-30, 1946

(Abstracts of Proceedings)

(Continued from the December 1946 issue)

Influence of Sulfonamides and Antibiotics on the Future of Otolaryngology

BERNARD J. MCMAHON, M.D.

ST. LOUIS, MO.

(This article appeared in full in the June issue, page 298)

DISCUSSION

DR. DEFOREST C. JARVIS, from his experience as secretary of a correspondence study group for over a period of 15 years, believes that we are approaching a rational application of biochemistry and physiology in the treatment of human ailments. He related his experience with a herd of cows among which there were cases of mastitis with streptococci. Adding a teaspoonful of cider for each 100 pounds of body weight to the feed resulted in the complete disappearance of the streptococci from the milk within three weeks. It was also noted that the odor of ammonia disappeared from the barn, showing that the putrefactive bacteria had been eliminated. By analogy, it was found that streptococcic sore throat and suppurative otitis media yielded promptly when cider vinegar was given the patient, thus changing the pH of the food. Giving the patient a teaspoonful of cider vinegar in a glass of water will often reduce the incidence of colds in a susceptible individual.

Dr. Thomas C. Galloway brought out the fact that frequently the administration of sulfonamides inhibits natural immunologic responses to infection, which frequently results in unresolved exudates that cause permanent harm as, for instance, in the ear after an otitis media. Here a plastic process may be the end result with permanent impairment of the hearing. In connection with adenitis Dr. Galloway finds that the employment of x-ray therapy will bring about absorption and resolution, whereas the sulfonamides will yield only a temporary slowing up of the process.

Dr. O. E. Van Alyea called attention to one of the dangers of uncontrolled sulfonamide therapy as recently brought out by some Chicago pathologists. It was shown in several cases at autopsy that a widespread endarteritis nodosa was present in the brain and heart, which could be explained on no other basis.

Dr. Frederick T. Hill commented on the fact that the biochemicals will not penetrate a fibrotic wall set up during an inflammatory process. He is at a loss to understand why penicillin introduced intrathecally is not recovered in the normal meninges but is found present in certain amounts when the meninges are inflamed.

Dr. William E. Grove pointed out a well-known fact, that the continued administration of sulfonamides frequently obscures the x-ray pictures of the bone, particularly the mastoid, so that accurate diagnosis of the progress of an infection cannot be determined from the reading of successive films.

Dr. John G. McLaurin reported having used penicillin solutions in the sinuses without finding it of much value in clearing up the infection. It is his impression that if one is to expect results from these drugs they should be given internally or subcutaneously in large doses for a comparatively short time and then discontinued if the desired results do not promptly ensue.

Dr. Terence Cawthorne is fairly committed to the use of antibiotics, but only if they are given the patient in a hospital where the symptoms and reactions can be closely watched and the therapy properly controlled.

Dr. Leroy A. Schall viewed the problem of antibiotics in relation to otolaryngology from the point of view of medical education. More training in pathology and bacteriology is necessary and our conception of the indications for operation will have to be revised. He feels that instead of contracting, otolaryngology must expand so that with proper training the newer otolaryngologists

will be prepared to treat allergy, malignancies and neurosurgical conditions arising out of ear, nose and throat conditions.

DR. FRED W. DIXON related two cases of apparently arrested mastoiditis under penicillin therapy, in which recurrence of symptoms and local signs of mastoiditis led to mastoid operations. In both cases the periosteum was found to be thickened and the cells filled with healthy granulations. No pus was present. He is at a loss to explain the situation but believes the operation might have been avoided had penicillin been given a third trial.

DR. MCMAHON, in closing, stated in reply to Dr. Galloway that the question of immunologic response to infection and the effect of antibiotics on this process is dependent in large measure on the type and degree of the infection. It is also possible that when there is an interval between two periods of therapy with antibiotics the body defenses may have an opportunity to mobilize in response to the infection during this interval, thus setting the stage for a more successful outcome from the second course of treatment. Concerning the use of x-rays in infection, Dr. MaMahon feels that whether antibiotics are given or not, the procedure should be helpful because it increases the blood supply to the infected area. For this reason the application of cold to an inflamed area, as the mastoid, for instance, tends to reduce the effectiveness of the antibiotics. As for giving sulfonamides to the patient at home, he makes it a practice to have the urine and the blood checked every few days so that possible ill effects may be recognized and properly treated. Also, he is in favor of giving adequate doses from the start until the desired effect is obtained or as long as the patient will tolerate the drug. Massive doses, however, may be dangerous because the sudden death of large numbers of bacteria may cause a fatal protein intoxication. This may explain a fatal meningitis in which the postmortem findings do not seem sufficient to account for the fatality.

Concerning the masking of the x-ray pictures, as mentioned by Dr. Grove, he feels that this condition presents a challenge to our diagnostic acumen. Our viewpoint on the interpretation of clinical signs must undergo revision and modification. This is corroborated by Dr. Cawthorne's statement relative to close observation of the patient in a hospital rather than haphazard treatment in the home. Dr. McMahon agrees with Dr. Schall on the necessity of broadening our knowledge in all related fields so that we have a thorough understanding of the patient as a whole.

He explained Dr. Dixon's cases on the redepositing of calcium in the areas where it had been previously absorbed. This would occur once the infection was halted and began to recede, and would explain changes observed in the x-ray pictures.

Neurogenic Tumors of the Nose and Throat

GORDON B. NEW, M.D.

ROCHESTER, MINN.

Tumors derived from nerve tissue or its coverings may be developmental, such as meningoceles, encephaloceles and hydroencephaloceles; or neoplastic, such as neurofibromas, ganglioneuromas and meningiomas. A variety of other terminology is cited. The pathogenesis and the histopathology of these various neoplasms is fully described.

In the past 27 years at the Mayo Clinic, 33 cases of neurogenic tumors have been encountered. There were 6 meningoceles, 3 encephaloceles, 19 neurofibromas, 1 ganglioneuroma and 4 meningiomas. Five of the meningoceles appeared at the root of the nose and one was intranasal. Most of them were pulsating and compressible. Diagnosis should be easy if the mass appears purplish in color and dents easily or if rhinorrhea is present. X-ray pictures may reveal the defect in the bone. It is dangerous to aspirate the mass and the problem is one for the neurosurgeon.

Encephaloceles appearing at the root of the nose may be mistaken for a dermoid. If there is no connection with the endocranium, removal is a simple task. The endonasal type is likely to have a direct stalk to the brain and must be attacked by the transantral or fronto-ethmoid approach.

The 19 neurofibromas were distributed about the pharynx, larynx and nose. Four were typically Recklinghausen's disease. They are slow growing and may be mistaken for mixed tumors, dermoids or lipoids. Brownish pigmentation of the skin or multiple tumors should suggest the correct diagnosis. They shell out easily, but in the pharynx bleeding may be troublesome. Preliminary tracheotomy and ligation of the external carotid may be indicated. Large tumors may necessitate an external approach.

Of the four meningiomas encountered, three were in the frontal sinus and one at the root of the nose. Absorption of bone and secondary hyperostosis may be present. Recurrence after surgery is possible unless the growth is thoroughly extirpated by surgical diathermy.

DISCUSSION

DR. PIERRE P. VIOLÉ recalled his report to this Society two years ago of a carotid body tumor in which the patient succumbed to a postoperative hemorrhage. Subsequently he encountered a schwannoma of the throat which was removed without any untoward effects. His thought in mentioning these cases was to emphasize the fact that preoperative diagnosis is difficult, and that one must be prepared for any contingency. Had the carotid artery been exposed under local anesthesia, ligation would have been possible in time, possibly, to save the patient's life.

DR. ROBERT C. MARTIN reported having seen two cases similar to the first one described by Dr. New, within a period of two weeks. Both were in infants. The first proved to be a simple cystic mass. The second, which had been diagnosed at birth as a meningioma, was subjected to the test described by Dr. Furstenberg, in which compression of the jugulars caused an expansion of the tumor. Fortunately this one also proved to be a dermoid cyst. It was dumbbell shaped with one lobe presenting externally to the nasal bones; this was connected by a neck which passed between the nasal bones to the other lobe which was expanded subperiosteally over the frontal bones. Its removal created a bony defect but the inner table was not affected.

The Problem of Secondary Frontal Sinus Surgery

FRANCIS L. WEILLE, M.D.

BOSTON, MASS.

(This paper appeared in full in the June issue, page 372)

DISCUSSION

DR. HARRIS P. MOSHER stated that frontal sinus surgery at his hands had been very disappointing. Temporary results have been common, but recurrences frequent. No operation could be termed successful under four or five years. It is his belief that failure is due to inadequate care of the infected ethmoid cells in series with

the frontal, and to closure of the enlarged nasofrontal passage. Recurrence and reoperation in one-third of the 276 cases reviewed by Dr. Weille does not speak well for the procedures employed. Secondary operations, as a rule, contemplated obliteration of the sinus and were successful only if this objective was attained. To truly obliterate the sinus it is necessary to remove both anterior and posterior walls as well as the floor. This should be less objectionable than formerly, since the use of tantalum for the correction of the cosmetic deformity has been proven successful.

Unless the recently projected methods of maintaining the patency of the new nasofrontal passage, such as Goodale's suspended strip of tantalum, Weille's tantalum tube or Hoople's acrylic mold prove definitely successful, there is no choice but a thorough obliteration of the sinus. Dr. Mosher cited Dr. Walsh's experiment, in which the nasofrontal duct was left alone as a matter well worth serious consideration. Dr. Mosher further suggested an obliterating operation in which the anterior and posterior walls are removed, but the floor is left untouched. The nasofrontal duct is left alone or only lightly curetted. The ethmoids are exenterated through the external exposure which permits the soft tissues to fill into the dead space. Naturally, the duct is obliterated. As mentioned before, the cosmetic defect is readily corrected by a tantalum implant.

Time alone will tell whether our dream of the perfect frontal sinus operation, in which the duct is permanently left open, will ever be realized.

Dr. W. L. Simpson agreed that exenteration of the ethmoids goes a long way toward preventing recurrence after a frontal sinus operation. In his experience, it has been the median cells which caused the most trouble and were most often overlooked. Complete removal of all of the frontal sinus mucosa is extremely important and, in extensively developed sinuses, may not be thoroughly accomplished. He has found it helpful to use a small laryngeal mirror in such cases to visualize the deeper recesses. He has had no luck with skin or mucosal grafts. As to the nasofrontal duct, Dr. Simson has given up trying to keep them open with tubes. He leaves the duct alone and finds that he has less trouble.

Dr. Anderson Hilding recalled some experiments he had made in the frontal sinuses of dogs, as well as those on the turkey's trachea, in which it was shown how a diaphragm will develop in the cavity as a result of concentric contraction of the scar which forms

in the denuded area. This explains some of the bad results necessitating secondary operation. He agrees with Dr. Walsh's experiment which showed how trauma of the mucosa of the duct leads to scar formation and obstruction.

DR. GEORGE L. TOBEY brought out the point that a healed sinus may become reinfected from a new upper respiratory infection, or that it is not always a case of recurrence. Corroborating Dr. Mosher's statement about the five-year period as a criterion of the permanence of the initial operation, Dr. Tobey cited two cases he had recently reoperated, one after 15 years and the other after 20 years. In both cases a mucocele was found. Scar tissue had obliterated a portion of the cavity, shutting off the rest from the nasofrontal passage. He agrees with Dr. Mosher that complete obliteration is the only certain method of obtaining a permanent cure.

DR. JONES (Washington) mentioned failures resulting from the use of vitallum tubes due to incrustation with calcium salts and referred to an article on this subject in the Western Journal of Surgery.

DR. WEILLE, in closing, referred to Dr. Mosher's statement regarding the removal of both walls of the sinus, leaving the floor intact. The records showed two patients operated on in this fashion, both of whom apparently did well. Commenting on Dr. Dixon's good results, Dr. Weille mentioned the fact that in the long series of cases which he had studied there were a number in which the operation was necessitated by the presence of a serious complication, such as meningitis, thrombosis, or brain abscess. In these cases, the mortality was high and in cases which recovered a secondary operation was necessary for obvious reasons. No doubt in this class of cases better results may be expected since the advent of intensive antibiotic therapy. He cited Allman, who had a record of 52 cases of orbital abscess or local infection, all of which cleared up under chemotherapy without surgery. Dr. Weille, like several of the speakers, has had no luck with skin grafts in maintaining patency of the tube. While he is impressed with Dr. Hilding's experiments and believes that the same mechanism is operative in humans as in dogs, he nevertheless has found a contrary point of view in Arey's experiments published in Physical Reviews in 1936, which do not coincide with Hilding's results. He suggests that possibly Dr. Hilding might revise Dr. Arey's views on this point. Referring to Dr. Tobey's experience with postoperative mucoceles, the records of his

series showed 48 mucoceles, of which 16 were postoperative, developing over an average of 9.2 years.

Dr. Weille has abandoned the use of vitallium tubes because of the difficulty of moulding them to the proper shape.

Congenital Cysts and Fistulae about the Head and Neck

CHARLES D. BLASSINGAME, M.D.

MEMPHIS, TENN.

DISCUSSION

DR. GORDON BERRY raised the question as to whether Carnoy's solution, which is made up of ferric chloride, alcohol and chloroform, had been tried in any of the cases. He had some good results in destroying residual fistulae with this solution where the processes were saccular rather than long tortuous passages, thus obviating the necessity of radical surgery with resulting scar formation.

DR. BLASSINGAME, in closing, stated that the concensus of the literature was against the use of escharotics in these cases. He feels that if the cyst or fistula is at all accessible it should be removed in toto.

Adamantinoma of the Maxillary Sinus

CLYDE A. HEATLY, M.D.

ROCHESTER, N. Y.

(This paper appeared in full in the June issue, page 322)

DISCUSSION

DR. JOHN J. SHEA showed some slides of an adamantinoma of the maxillary sinus, proven by biopsy, in a woman who was five months pregnant. She was not seen until two years later when the picture was apparently unchanged. She was again pregnant and operation was again deferred.

DR. THOMAS C. GALLOWAY emphasized the fact that though these tumors grow slowly and seem harmless, they are insidious and extend along the periosteum to involve remote areas. His experience in three cases has taught him to be thorough in the surgical technic so that no trace of the growth be left.

Abstracts of Current Articles

EAR

Audiometer Investigations with Sinusoidal Waves from 16 Cycles to 20,000 Cycles.

Juul, A.: Acta Oto-laryngologica 31:386, 1943.

The author describes an audiometer using sinusoidal sound waves within a frequency range of from 16 to 20,000 cycles. An investigation was conducted as to the effect of the injections of sodium salicylate upon hearing. It was found that this caused a reduction in hearing in the upper frequencies by both air and bone conduction, presumably due to the toxic effect upon the spiral ganglion or at some other point central to the organ of Corti.

HILL.

Rubella As A Cause of Congenital Deafness In England.

Clayton-Jones, E.: The Lancet 152:56-61 (Jan. 11) 1947.

In 1943 and 1944 Swan and his associates found, during an epidemic of rubella in South Australia, that of 101 children whose mothers had rubella during pregnancy 78 were born with various congenital defects, mainly cataracts, deaf-mutism and heart lesions. He also found that of the 78 mothers with defective children 69 had the rubella in the first three months of pregnancy, 8 in the fourth, fifth and sixth months and only one in the last three months. They concluded that if the mother contracts rubella in the first two months the chances of her child being born defective are about 100 per cent, if she contracts the exanthem in the third month the chances are about 50 per cent, whereas later in pregnancy the chances are very low.

The virulence of the disease may have some bearing on these pessimistic statistics because during a 1946 epidemic of rubella in Milwaukee, Fox and Bortin found only two congenital abnormalities in 12 babies whose 11 mothers had rubella during pregnancy, 5 in the first two months, 4 in the third or fourth month and 1 in the seventh month. There was one set of twins.

Deafness is not easily diagnosed in very young babies but Swan and his associates found 38 cases of deafness (4 doubtful) among

his 78 children with congenital defects. As to the relation between the stage of pregnancy and the type of malformation in the child, Swan and his colleagues found that the average duration of pregnancy at the time of the rubella attack in the mothers of children with cataracts was 1.4 months, in mothers of children with heart lesions, 1.5 months and in mothers of deaf children it was 2.3 months.

In the cases thus far reported from the United States, deaf-mutism is an unusual finding but Jones suggests that this may be due to early reporting as most of the cases published have been of children under two years of age.

Clayton-Jones made a survey in three large schools for the deaf at Manchester, Doncaster and Birmingham and found by questionnaire 11 children among 123 whose mothers had had rubella during pregnancy, or 8.9 per cent. At this same time a similar survey made by a Miss Martin in her speech therapy clinic and various schools for the deaf throughout England revealed a total of 102 deaf children born in England during the rubella epidemic of 1940-41. She obtained a history of rubella during the first four months of pregnancy in 36 of the mothers of these 102 cases, a percentage of 35.

Clayton-Jones reports that the deafness in all cases where it could be tested was of the perception type and its degree usually the same in the two ears. The deafness is rarely, if ever, complete. In an infant whose mother had rubella during the first month of pregnancy and who died at the age of 6½ months with congenital cataracts and a patent ductus arteriosus, the outstanding feature of the inner ear at autopsy was the lack of differentiation of the primitive cells to form the organ of Corti. The vestibular end organs showed the same lack of differentiation as the cochlea.

According to the dictate of Mann (quoted) that the rubella virus only affects organs which are not yet fully developed in the fetus, Clayton-Jones states that the most rapid differentiation of the cochlea is taking place at about the time of the seventh week and that when rubella attacks the pregnant mother at about this time, this organ is apt to cease differentiation.

The author implies that the discrepancy in the reported percentages of congenital defects in children of mothers who contract rubella during pregnancy may be in some measure due to the variation in the virulence of the disease during different epidemics.

GROVE.

The Fenestration Operation For Otoslcerosis.

Hall, Simon: *Brit. M. J.*, Nov. 2, 1946, pp. 647-649.

Hall outlines the historical development of the operation. The principle of the operation is to restore the recoil action of the intracochlear fluids. He lays great stress on the proper selection of cases indicating that the best results may be expected in young individuals with a moderate loss of hearing. Increasing age introduces other factors which tend to reduce the possibility of restoration of hearing but he maintains that the operation should not be denied to these older individuals merely to improve the operator's statistics. He also believes that in certain of the older patients the progress of the hearing deterioration is stopped even if the hearing does not improve above the preoperative level. For the best results the patient should be young, there should be no sign or history of middle ear disease, there should be no nerve deafness and the hearing loss within the speech range should not exceed 40 to 50 decibels.

Hall does his operation under either local or general anesthesia. He outlines the steps of his operation which can be performed either from a retroaural approach or by an endaural approach. He states that signs of labyrinthine disturbance are immediate after opening the labyrinth and may be severe but are not liable to persist for more than three days. He notes that some patients are confused by the postoperative volume of sound and says that they may have to be reeducated to understand speech under the new postoperative conditions.

Under the causes of failure he lists the selection of unsuitable cases, failure in technique, presence of previously undiagnosed disease, otitis media and a gradual obliteration of the fistula. He has not been able to confirm the statement that the use of the cartilaginous stoppel has lessened the postoperative loss of hearing.

The total number of operations performed by Hall was over 200. Of these only 118 consecutive cases were selected for this report. This series excludes the earliest ones and also all operations performed in 1946. A minimum of eight months was allowed to elapse before the case was counted. Of the 118 cases, 82 patients are still improved, a percentage of 69. A breakdown of these statistics according to the age of the patient gives 80 per cent improvement in patients under 19 years, 77 per cent of those under 24 years, 62 per cent of those under 34 years and 61 per cent of those under 39 years. This demonstrates the diminishing probability of success as age increases.

Hall had one case of facial paralysis which recovered completely; no cases of labyrinthitis and two fatalities. One of these was due to an acute cardiac failure and the other to morphine poisoning.

GROVE.

NOSE

The Surgical Treatment of Chronic Frontal Sinusitis.

Negus, V. E.: Brit. M. J., Jan. 25, 1947, pp. 135-136.

In dealing with chronic sinusitis of the frontal sinuses, Negus stresses two points. First, it is necessary to eliminate all contributory factors in the nasal fossa. Among these are infection in the maxillary sinus and the ethmoid cells which should first be cleared up, high deviations of the septum which block drainage, and enlarged and obstructing middle turbinates which can do the same thing.

The second point concerns the direct treatment of the infected frontal sinus after all preliminary measures have been taken to insure an adequate outlet to the nasofrontal duct. Negus does not believe that intranasal operations on the nasofrontal duct are either indicated or warranted, particularly because of the tendency of the duct to close after such intranasal manipulation.

Negus believes that if an adequate nasofrontal duct is provided the frontal sinus will in the majority of cases return to normal provided that its lining mucosa has not been disturbed. He has had good results with a number of cases in which an obliterating operation had been unsuccessfully employed by establishing an adequate nasofrontal duct. He does not believe that curettage of the lining of the sinus is necessary or advisable. He states that it will return to normal if proper ventilation is provided. He makes his incision around the margin of the orbit well away from the inner canthus. He says it is unnecessary to remove much of the floor of the sinus but it is necessary to remove a considerable portion of the nasal process of the maxilla. Exuberant hyperplastic mucosa is gently removed when necessary but curettage is carefully avoided. He does not believe that an examination of the posterior wall for caries is necessary for the reason that even if it is present it had better be left alone.

In order to maintain patency of the nasofrontal duct he uses a skin graft applied to the raw orbital periosteum and to the exposed

margin of the bone at the entrance of the frontal sinus. The graft is tucked into position after the insertion of a large tube of rubber or plastic material. It is not wrapped around the tube. The tube is removed in ten days and no subsequent dilatation or sounding is done.

GROVE.

Method of Counting Eosinophil Cells in the Nasal Secretion of Patients Suffering From Allergic Rhinitis.

Kragh, J., and Norgaard, A.: Acta Oto-laryngologica 31:445, 1943.

The authors have endeavored to develop a method of counting cells in nasal secretion similar to the differential blood count. The difficulty of uneven distribution is overcome by homogenization by drawing the secretion in and out of a graduated syringe. If very mucoid, the secretion may be diluted by a measured amount of a weak buffer solution.

HILL.

PHARYNX

Lymphoepithelioma.

Tice, Galen M.: Radiology 47:116-127 (Aug.) 1946.

Fifty-one references are cited and 12 cases discussed to emphasize the uncommonness of, but distinct clinical and histologic features of, lymphoepithelioma of the nasopharynx. Intensive radiation therapy should be started as soon as the diagnosis is made. Radium and x-ray therapy should be used alone or in combination, depending upon the facilities available and the accessibility of the tumor. Evidence seems to the author to favor a specific tumor called lymphoepithelioma. Whether transitional cell carcinoma, undifferentiated cell carcinoma, and lymphoepithelioma are essentially the same histologically has not been definitely decided. For therapeutic purposes the matter is of academic interest.

JORSTAD.

MISCELLANEOUS

Congenital Cystic Disease of the Lung.

Savain, William, and Bryant, J. E.: Radiology 47:156-162 (Aug.) 1946.

Three cases of congenital cystic disease of the lung are reviewed and seven references cited. Cystic disease proper, called also con-

genital bronchiectasis, occurs mostly in adults. Treatment offers a favorable prognosis. The second type, or "fluid cyst", may simulate a pleural effusion or, after the cyst contents are evacuated, a pneumocyst. Infection and intrathoracic pressure changes are the most serious complications. Prognosis is better in adults than in children. Treatment consists in supportive measures, thorocentesis, permanent drainage, and lung resection.

JORSTAD.

The Cough Syrup.

Boyd, Eldon M.: Brit. M. J., Nov. 16, 1946, pp. 735-736.

Boyd, of the Department of Pharmocology, Queen's University, Kingston, Canada, attempted to determine whether the vehicle used in the common cough syrups exerted any anti-tussive action per se or if the anti-tussive action of cough syrups was due solely to the expectorant dissolved in the syrup.

The technique used was to measure the output of the respiratory tract fluid in experimental animals by ligating a T-shaped cannula into the trachea of lightly anesthetized or decerebrate animals (rabbits, cats and guinea pigs). The tracheobronchial secretions were collected and measured over a two-hour period before the administration of any medication. After this initial period expectorant medication was introduced into the stomach via a stomach tube and the hourly amount of tracheobronchial secretion again measured.

From the results of these experiments on animals and also from the results of experiments on man, Boyd comes to the following conclusions: First, the syrupy vehicle of a cough syrup exerts a short demulcent effect upon the mucosa of the pharynx and upper respiratory tract and if the cough is due to irritation of this mucosa, the syrupy vehicle has an anti-tussive effect per se. Secondly, when the syrup enters the stomach it has little or no expectorant property in itself in that it does not cause any increase in the tracheobronchial secretions. Third, effective expectorant drugs added to the syrup begin to increase the output of tracheobronchial fluid at about the time that the local anti-tussive action of the syrup alone has begun to wear off. Boyd believes that these experiments offer a logical explanation for the time honoured use of expectorant drugs in a syrupy vehicle.

GROVE.

Penicillin Inhalation Therapy.

Segal, M. S., and Ryder, C. M.: New England J. Med. 236:132 (Jan. 23) 1947.

Eighty-five patients with pneumonia, infective bronchitis, infective bronchial asthma, lung abscess, bronchiectasis, infective laryngo-tracheo-bronchial edema, pulmonary emphysema and pulmonary blebs were treated with penicillin aerosol with considerable improvement in many cases. Peak blood levels were observed at 30 minutes and in some cases persisted for two hours. The authors do not condone this treatment for self-limited conditions, such as the common cold, warning against the possibility of the appearance of penicillin-resistive strains in all cases of repeated or prolonged penicillin aerosol therapy. Every attempt should be made to identify the causative organism. Penicillin may be safely combined with streptomycin aerosol when indicated.

HILL.

Notices

UNIVERSITY OF CINCINNATI

The Eleventh Annual Graduate Convention in Otology, Rhinology and Laryngology will be held at the University of Cincinnati College of Medicine, April 14 to April 19, inclusive, 1947. It will consist of an operative course on the cadaver and didactic lectures.

STATE UNIVERSITY OF IOWA

During the 1947 Summer Session the Departments of Speech, Psychology and Otolaryngology of the State University of Iowa are again offering an intensive four weeks' course in Audiometry and the Fitting of Hearing Aids. The course will run from June 23 to July 19 and is open to anyone meeting college entrance requirements. A series of weekend lectures and round table discussions on various aspects of hearing conservation will be featured as a part of the summer program. Inquiries may be addressed to Jacqueline Keaster, Assistant Professor, Speech and Otology, The Speech Clinic, State University of Iowa, Iowa City, Iowa.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct examinations at the Coronado Hotel, St. Louis, Missouri, April 16 to 19, 1947, and at the Palmer House, Chicago, Illinois, October 7 to 11, 1947. Communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

BACK ISSUES OF THE ANNALS

The Annals is desirous of obtaining copies of the following issues now out of print: March 1940, March 1943, June, 1943, September 1943, December 1943, March 1944, December 1944, March 1945, June 1945, September 1945, and March 1946. $1.50 per copy will be paid for any of these issues returned to us. They should be sent with a memorandum of the sender's name and address to the Annals Publishing Company, 7200 Wydown Blvd., St. Louis 5, Missouri.

HOME STUDY COURSES

The Home Study Courses sponsored by the American Academy of Ophthalmology and Otolaryngology, in the basic sciences of those two specialties, will be given again beginning September 1, 1947. Registrations must be completed before August 15. Detailed information may be secured from Dr. William L. Benedict, Executive Secretary, 100 First Avenue Building, Rochester, Minnesota.